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THE JOURNAL

OF THE SOUTH CAROLINA MEDICAL ASSOCIATION

SHELVE UPDT

EMBOLIZING CARDIAC MYXOMA

MUCOCUTANEOUS LYMPH NODE SYNDROME

THE INFECTION PRONE PATIENT

TEACHING HUMAN VALUES TO MEDICAL STUDENTS

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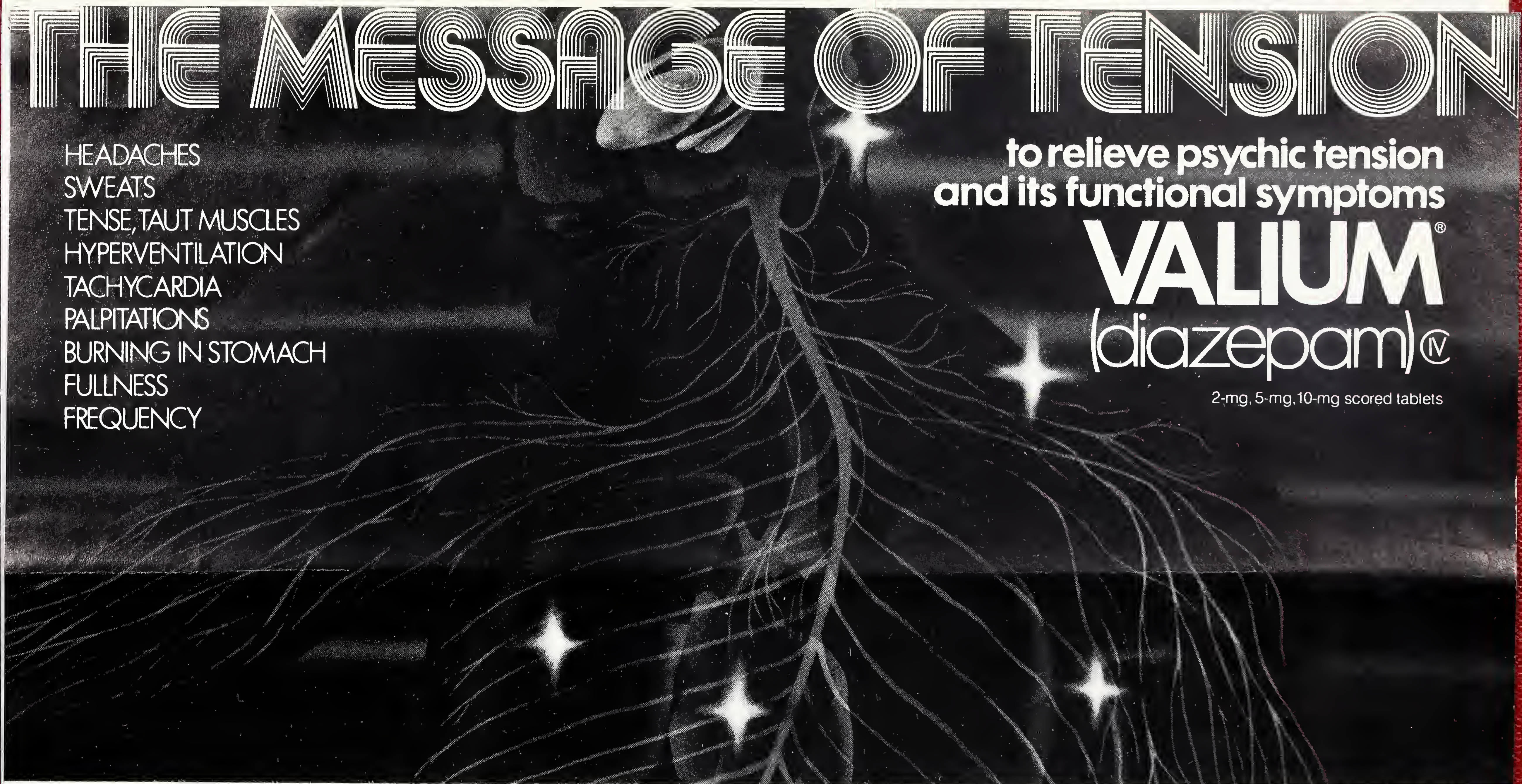

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AORTIC OBSTRUCTION FROM EMBOLIZING CARDIAC MYXOMA*

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The myxoma accounts for more than 50% of intracardiac tumors. It is three times more frequent in the left atrium than in the right and occasionally occurs in the ventricles. Untreated, they are potentially lethal.

Crafford[3] reported the first successful removal of a myxoma in 1955. Presently, curative resections with modern open heart techniques are the rule rather than the exception. In removing these tumors, symptoms usually abate dramatically postoperatively. Diagnosis is often difficult and delayed because of the protean manifestations of these tumors. Some remain asymptomatic until a catastrophic event occurs.

We encountered the case of an otherwise healthy patient who sustained aortic and iliac artery obstruction with sudden embolization from a left atrial myxoma. The pathological material removed was diagnostic and the patient subsequently had a successful resection.

CASE REPORT

W. F. was a 43 year old white female who had experienced sudden weakness in both lower extremities three months prior to admission. Since the initial onset, she noted progressive claudication of both lower extremities. She was a nonsmoker and had no history of diabetes, heart disease, or atherosclerosis. Physical examination revealed absent right femoral and pedal pulses and left pedal pulses. Laboratory values were within normal limits. An aortogram revealed a 60% occlusion of the left common iliac artery and a complete occlusion of the right iliac artery. A saddle embolus was suspected by the radiologist. (Figure 1)

On 6/25/75, an aortoiliac thrombectomy was done by H. G. with closure by patch graft to the terminal aorta. The material removed from the aorta was gelatinoid in consistency. The arterial walls did not appear abnormal. Malignant cells were demonstrated in the clot removed from the aorta suggesting an intracardiac tumor. A thorough investigation excluded other organ malignancies. An echocardiogram suggested, and a subsequent angiocardiogram confirmed, the presence of a left atrial tumor. The tumor protruded through the mitral valve during ventricular diastole and withdrew into the atrium during the ventricular systole.

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On 7/10/75, removal of a left atrial tumor was accomplished by R. B. and H. G. through a left atriotomy with extracorporeal circulation. The tumor mass was large, had a gelatinous consistency similar to frog eggs, and fragmented easily. It was attached to the outer edge of the atrial septum by a short narrow stalk. The base of the stalk was removed with an ellipse of the atrial septum. Great care was taken to prevent fragmentation and embolization. The atrium and left ventricle were irrigated copiously. The aorta was maintained cross clamped. The right atrium was opened and inspected for tumor but none was found. She recovered satisfactorily and has done well since.

A pathologist confirmed the diagnosis of myxoma and found it to be similar to the material removed from the aorta previously.

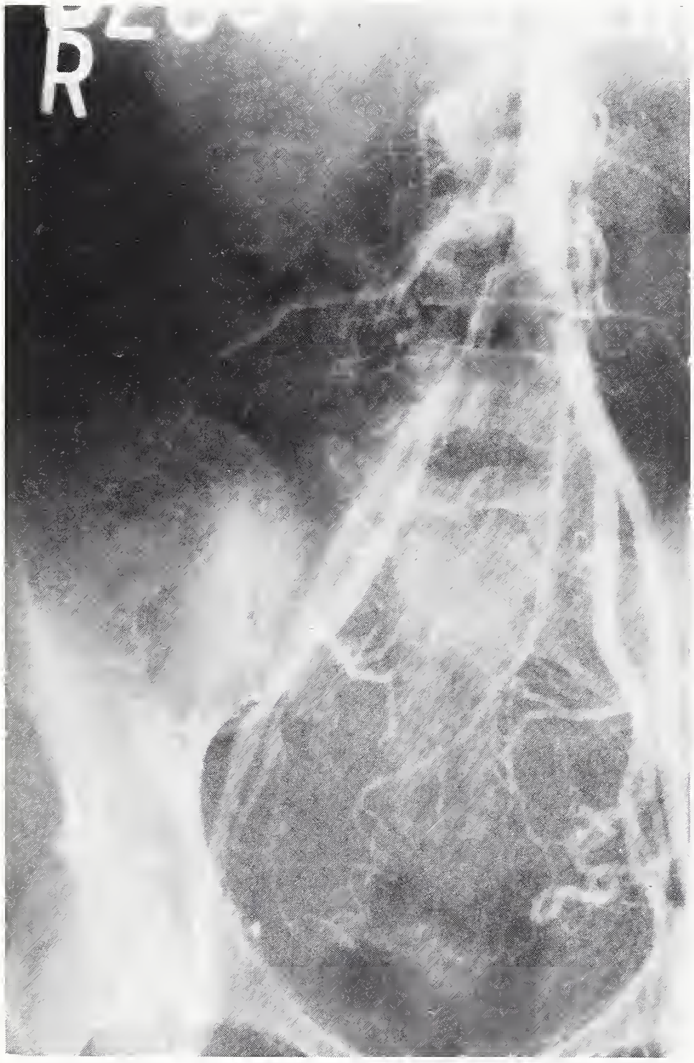

FIGURE 1: Aortogram demonstrates a saddle embolus with 60 percent occlusion of the left common iliac artery and near-complete occlusion of the right common iliac artery.

DISCUSSION

Cardiac myxomas are usually found in the atria but occasionally occur in the ventricles. Of eight myxomas reported by Symbas and Associates,[14] six were in the left atrium and two were in the right atrium. Collins and Collins[2] found four in the left atrium, three in the right atrium, and one in the left ventricle. Of thirteen patients with myxoma reported by Hattler and colleagues,[6] ten were in the left atrium and three were in the right atrium. Anderson and associates[1] reported a patient who had biatrial myxomas and underwent successful removal of both simultaneously.

A familial tendency is rare. However, Lieblew and his group[9] documented four siblings with myxomas, three of these children having two or more. One sibling had four myxomas excised.

Recurrent myxomas have been reported. These occur by (1) embolization and growth, (2) incomplete excision and regrowth of the original tumor, (3) intracardiac implantation, and (4) growth of a second focus.[4]

Sections of the tumor that we removed at operation revealed a myxoid mass with deposits of fibrin both within its substance and on its surface. The myxoid material was vascularized and contained a considerable number of cells which varied from spindle and branching elements to multinucleated forms. Some of the cells were arranged in rows and frequently paralleled the vasculature. It was the same type of multinucleated cellular form observed in the thrombo-embolus previously.

It is believed that myxomas develop from primitive endothelial or subendocardial cells that are found more abundantly in the region of the fossa ovalis. This might explain some of the recurrences of a tumor that has been totally excised.[8]

The manifestations of myxomas vary greatly due to embolization to the systemic and pulmonary systems, obstruction to blood flow through a cardiac chamber or valve, or to certain constitutional symptoms. Many of these tumors go unrecognized for long periods of time. Some are misdiagnosed as rheumatic heart disease with mitral stenosis, myocardiopathy, bacterial endocarditis, pulmonary embolus, and congestive heart failure.

Three of six patients reported by Mundth and associates[11] presented with peripheral vascular emboli. The discussion of their cases cites instances of emboli to the aortic bifurcation, the abdominal aorta, the renal arteries, the cerebral arteries, and the popliteal arteries. Certainly, the frequency of emboli promotes urgency in removal once the presence of the myxoma is confirmed.

The myxoma in our patient, although large and although protruding through the mitral valve with each heart beat, did not produce any significant obstruction to blood flow. This can occur by virtue of size with displacement of blood in the cavity or by obstructing a valve as it protrudes through it. Holswade and associates[7] reported a case in which successful removal of a myxoma was performed after two years of treatment for congestive failure. Finally, angiocardiography demonstrated the characteristic filling defect and prolapse of the tumor from the right atrium into the ventricle. Rose and associates[13] reported an unusual case of a left atrial myxoma traversing the mitral valve orifice and reaching a point just below the aortic valve. Mild aortic regurgitation was noted. At operation, the left and non coronary cusps were noted to be shrunken and fibrotic but the right cusp appeared normal which suggested that the aortic regurgitation was secondary to impact from the myxoma against the aortic valve. Two of the eight patients with right atrial myxomas reported by Collins and Collins[2] had severe tricuspid valve insufficiency presumably due to traumatic impingement of the tumor on the valve — the so called "wrecking ball" effect. In their cases, the tumor obstruction of the mitral valve was found to be indistinguishable from mitral valve stenosis and the tricuspid valve obstruction was found to be similar to constrictive pericarditis with ascites and decreased cardiac output.

Goodwin[5] reported 89% incidence of one of the following constitutional symptoms: fever, increased erythrocyte sedimentation rate, weight loss, anemia, or abnormal serum proteins. May and associates[10] reported that the constitutional symptoms often mimic bacterial endocarditis and so called "collagen disease." They attribute the postural syncope, changing murmurs, arrhythmias, and sudden episodes of dyspnea alternating with periods of apparent good health, to mechanical obstruction to blood flow. These authors also gave an interesting comparison of clinical, electrocardiographic, radiographic, and hemodynamic findings in left atrial myxoma and in mitral stenosis (from the literature).

One of the most important factors in diagnosis is to remain aware of cardiac myxomas and include them in the differential diagnosis of such diseases as rheumatic heart disease and bacterial endocarditis. Another important factor is the pathological examination of all emboli or thrombi, especially when sudden obstruction of a vessel occurs or where pulmonary emboli occur in an otherwise healthy patient. Phonocardiography may demonstrate a questionable "tumor plop" and strengthen the suspicion of a myxoma, especially a left atrial one. Echocardiography is a good screening process. Angiocardiography is essential for verification. Injections of contrast material into the superior vena cava and pulmonary artery should be done to demonstrate right and left side myxomas respectively. Hattler and associates[6] stressed that electrophoretic changes in the serum proteins with increase in the gamma globulins are features of diagnostic importance and might be ascribed to an immunological reaction to the tumor. They found that the serum globulin pattern consistently returns to normal following removal of the myxoma and with subsidence of symptoms.

Removal of the tumor has been uniformly successful in the majority of the patients and should be done as soon as feasible after the diagnosis is established. To quote one author,[10] "these patients are literally walking a tight rope between calamitous systemic embolization, intractable cardiac failure, and sudden death." Repeated embolization in two young patients reported by Collins and Collins[2] produced permanent neurologic deficits in each.

All operations should be performed with cardiopulmonary by-pass. Because of the friability of these myxomas, the heart should be defibrillated and the aorta or pulmonary artery cross clamped to avoid embolization secondary to fragmentation. Both the left and right atria should be opened and examined. The ventricles can be examined through the tricuspid and mitral valves. The tumor must be extracted in a gentle manner to avoid fragmentation. The atrial cavity and contiguous ventricle should be thoroughly irrigated to remove any fragments. At least two fine mesh filters should be used in the arterial line and one coarse filter used in the venous line. Trinkle, Edelsteen, and Yoshonis[15] found tumor emboli present in the second 50-mesh filter in the arterial line, which passed through one coarse venous line filter and the first 50-mesh arterial line filter. The portion of the septum to which the pedicle is attached should be excised. Kabbani and Cooley[8] advocate excision of most of the atrial septum (including the entire fossa ovalis region) in continuity with the tumor so as to provide better protection against recurrence of this type of neoplasm.

The results with operative excision of cardiac myxomas have been excellent as was the case with our patient. Relief of symptoms is dramatic. Recurrences are rare. There remain three important concepts, however. The first is to keep cardiac myxomas in mind and include them in the differential diagnosis of the conditions that they can mimic. The second is to have pathological examination of all embolic material extracted. The third is to follow these patients for a long period of time postoperatively remaining sensitive to recurrences. □

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MUCOCUTANEOUS LYMPH NODE SYNDROME IN SOUTH CAROLINA*

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Mucocutaneous lymph node syndrome (MLNS), an acute febrile illness of unknown etiology, is being identified with increasing frequency in the continental United States. The pattern of spiking fevers, distinctive exanthem and enanthem, widespread tissue inflammation of viscera, heart and central nervous system, and unresponsiveness to antibiotic therapy is sufficiently characteristic to permit diagnosis on clinical grounds alone. Since the initial description by Kawasaki in Japan in 1967, more than 7,000 cases have been observed in that country.[1] The disease was first reported in the continental United States in 1974.[2] Only one case of MLNS has been reported from South Carolina.[3] During the past two years, we have observed four children with this disease. Experience with these children provides the basis for this report.

CASE REPORT

D. C., a 6 year old white male was admitted to Anderson Memorial Hospital following an 18 hour history of fever, vomiting and abdominal pain. Prior to the onset of these symptoms, he had been in excellent health. He presented a generally toxic appearance with heart rate of 120 beats per minute, respirations 44 per minute and temperature of 40°C. There was a widespread exanthem consisting of bright erythema of the lips, erythematous papules on the trunk with accentuation in the axillae and inguinal areas, and erythematous macules on the palms and soles. There was injection of the conjunctivae bilaterally, and a bright erythema of the oral mucosa, pharynx, and tongue. Prominent papillae gave the tongue a strawberry appearance. The neck was supple, and tender anterior cervical lymph nodes measuring 1-2 cm. in diameter were present. The initial cardiopulmonary examination was normal, except for increased heart and respiratory rates. Abdominal examination showed slight tenderness to deep palpation.

Laboratory findings included a leukocyte count of 26,200 with 80% mature neutrophils and 12% band forms, platelet count of 1,160,000 and erythrocyte sedimentation rate of 53 mm per hour. Döhle bodies and toxic granulations were present on the peripheral blood smear. The urine contained protein and bilirubin. Serum chemistries included: protein 5.1 gm/dl, albumin 2.5 gm/dl, alpha 1 globulin 0.5 gm/dl, alpha 2 globulin 1 gm/dl, bilirubin 3.5 mg/dl with predominance of the direct fraction, SGOT 110 mu/ml, and prothrombin time of 58.3% activity. Serologic studies showed immunoglobulin E of 22 units/ml, positive C-reaction protein, antistreptolysin-O titer of 50 Todd units, negative mono spot test, negative Australia antigen, cold agglutinin titer of 1:8, and positive febrile agglutinins with salmonella OX-K, and OX-2 titers of 1:20. The throat culture had only normal flora and cultures of the urine and blood were negative. Stool culture grew *Staphylococcus aureus*, coagulase positive. The initial electrocardiogram showed sinus tachycardia, the chest x-ray was normal, and barium enema was normal with filling of the appendix.

After appropriate cultures were taken, treatment was initiated with one million units of aqueous Penicillin G administered intravenously every 6 hours. This was discontinued after report of the negative throat culture.

The hospital course was marked by high spiking fevers, mild diarrhea, accentuation of the erythema affecting the palms and soles, and edema of the distal extremities. The tachypnea increased and chest x-ray on the 5th day of hospitalization showed bilateral perihilar pulmonary infiltrates which were treated with ampicillin and oxacillin intravenously. Digoxin and furosemide were added on day 7 because of cardiac decompensation, evidenced by increasing tachypnea, enlarging liver, and x-ray evidence of cardiomegaly. An electrocardiogram at that time showed right ventricular strain. These therapeutic measures produced no observable improvement. Gradual clinical improvement began on the 8th hospital day, coincident with the onset of desquamation of the skin beginning at the junction of the digits and nailtips. Fever and other symptoms had completely resolved by the time of discharge on the 16th day. Examination and electrocardiogram at 4 weeks following discharge have been normal.

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The findings in this case and the three other cases are summarized in Table 1.

DISCUSSION

Characteristic clinical manifestations of mucocutaneous lymph node syndrome include: (1) fever lasting from 1-2 weeks, (2) congestion of the conjunctivae, (3) erythematous tongue and oropharyngeal mucosa with red fissured lips, (4) erythema of the palms and soles, (5) indurated edema of the hands and feet, (6) polymorphous generalized rash, (7) non-suppurative cervical lymphadenopathy, and (8) evidence of widespread inflammation involving viscera, brain and heart. Arthritis or arthralgia, diarrhea, abdominal pain and mild jaundice may occur. A characteristic desquamation occurs during the second week, beginning at the free margin of the nails. A shedding of the nails may be noted during the weeks following the disease.[4, 5] This phenomenon occurred in one of our patients. The disease predominately affects young children. The accompanying laboratory findings include leukocytosis with predominance of neutrophils, thrombocytosis, increased sedimentation rate, in-

Table 1

Features in Four Cases of MLNS in South Carolina

	case			
Principal Manifestations	1	2	3	4
age	6 yr	4 yr	6 yr	9 mo
duration of fever	15 days	19 days	13 days	13 days
conjunctval injection	yes	yes	no	no
changes of lips and oral cavity	yes	yes	yes	yes
redness and edema of hands and feet	yes	yes	yes	yes
desquamation from fingertips	yes	yes	yes	yes
cervical adenitis	yes	yes	yes	no
Associated Findings				
cardiovascular signs	yes	no	no	yes
proteinuria or pyuria	yes	yes	yes	yes
diarrhea	yes	no	no	no
arthralgias	no	no	no	?
aseptic meningitis	not done	not done	not done	yes
jaundice or increased serum transaminase	yes	yes	yes	
leukocytosis with left shift	yes	yes	yes	yes
increased ESR	yes	yes	not done	yes
anemia	yes	yes	no	no
positive C-reactive protein	yes	yes	not done	no

creased transaminases, negative ASO titer and other evidence of streptococcal infection, increased alpha-2 globulins and a sterile pyuria.[6]

Although the prognosis for most affected children is good, the 1-2 percent mortality makes mucocutaneous lymph node syndrome a significant disease. The mortality is associated with sudden death from myocardial infarction or arrhythmia. Death usually occurs during the third or fourth week of illness but may occur later.[7] Coronary arteritis with aneurysms or thrombosis and papillary muscle dysfunction with mitral valve incompetence have been seen at autopsy.[1] Nonfatal cases have a high incidence of abnormal electrocardiograph findings (90%) and abnormal coronary angiograms (60%).[8, 9] It has been speculated that patients with persistently elevated IgE levels may be more susceptible to serious cardiovascular complications.[10] Some investigators have suggested that coronary angiography should be performed on all survivors who have had evidence of significant cardiac involvement.[7]

The treatment of mucocutaneous lymph node syndrome has been symptomatic. Antibiotics have been ineffective. A recent report indicates that treatment with large doses of corticosteroids (Prednisone 3 mg/kg/day) and/or aspirin (150-200 mg/kg/day) will result in dramatic response.[11] The most appropriate therapeutic measures must await further clinical trials.

The cause of mucocutaneous lymph node syndrome is not known. The findings in MLNS suggest an infectious etiology but cultures and serological investigations have not found any infectious agents.[12] Rickettsia-like bodies were reported by electron microscopy in skin and cervical lymph nodes in 12 of 23 patients with MLNS studied by Hamashima.[13, 14] These findings have not been found in other investigations. Despite the similarities with Stevens-Johnson syndrome

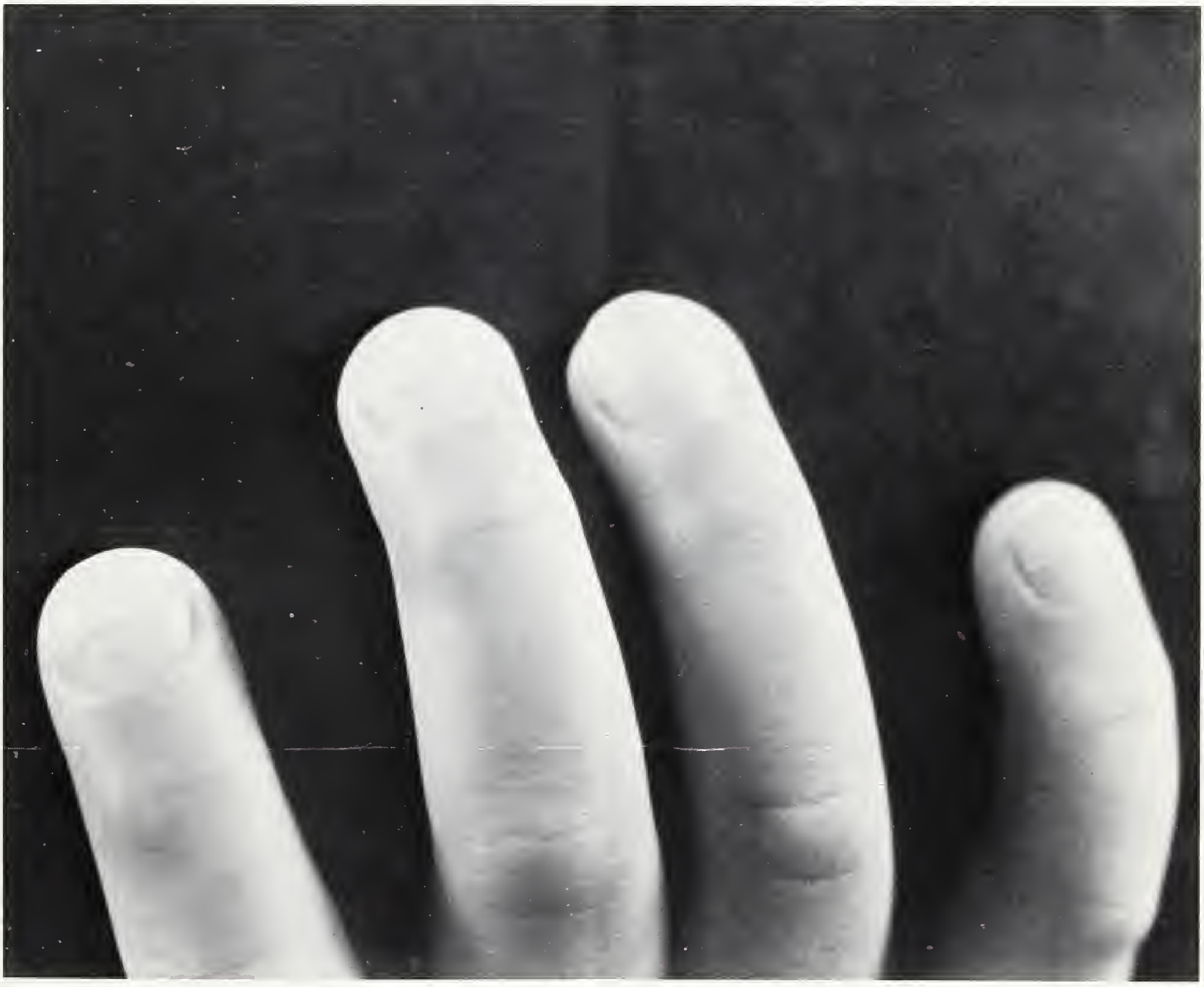

FIGURE 1. Desquamation beginning at the free margin of the nails occurs during the second week of illness.

and infantile polyarteritis nodosa, there has been no biological evidence to support a hypersensitivity reaction in patients with MLNS.[14, 15] Likewise, no exposure to toxic agents has been documented.

The differential diagnosis includes a number of entities. There are many similarities to scarlet fever, a diagnosis which can be ruled out by the prolonged febrile course, absence of culture and serologic evidence of streptococcal infection, and failure to respond to streptococcal sensitive antibiotics. Systemic juvenile rheumatoid arthritis can present similar clinical findings but has less dramatic erythema of the palms, soles, and lips, a more salmon pink rash, and no myocardial or meningeal inflammation. The Stevens-Johnson syndrome, likewise, has symptoms similar to MLNS, but its bullous exfoliative dermatitis with oral ulcerations and purulent conjunctivitis serve as differentiating features. The pathologic features seen in fatal cases of MLNS resemble those of infantile polyarteritis nodosa, especially as they relate to coronary artery involvement. Mucocutaneous lymph node syndrome, however, unlike infantile periarteritis nodosa, is a self-limited disorder usually resolving within a matter of several weeks.[15, 16] Other entities such as Rocky Mountain Spotted Fever, infectious mononucleosis, coxsackie infections, and Henoch-Schönlein purpura may likewise be confused with MLNS.

The diagnosis of MLNS remains a clinical one, but one supported by principal features which are distinct and easily recognizable. The treatment is supportive with cautious observation for cardiovascular complications. Limited experience indicates that massive corticosteroid and aspirin treatment may be beneficial. The disease is usually self-limited with the mortality related to cardiovascular complications. ☐

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MANAGEMENT OF THE INFECTION PRONE PATIENT: A GUIDE FOR THE FAMILY PHYSICIAN*

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GEORGE G. DURST, JR., M.D.

Immunological reactions have been known to play a role in resistance to bacterial infections for three quarters of a century, but it was not until 1952, when Burton discovered hypogammaglobulinanemia, that defects in immune defense were proven to be associated with an increased susceptibility to infection in man.[1] Since that time a large variety of inherited abnormalities of the immune process have been studied. Such studies have led to a better understanding of the precise role in antimicrobial defense. These studies in turn have generated interest in documented acquired defects of immunity associated with physiological changes, disease, or the treatment of disease. Acquired defects are now known to be numerous and they frequently contribute to development of infections in the hospitalized patient. In fact, most, if not all, patients considered to be infection prone can now be assumed to have abnormalities of host defense. Identification and characterization of such abnormalities, however, remain difficult because of the incompletely understood, complex interactions within the immune system and the uncertainty of the clinical relevance of many immune system components. In addition, many of the tests available to the research microbiologist and clinician are not readily available to the family physician or internist in the general medical community. An attempt will be made to identify the tests for detecting defects in immunity and which appropriate actions may be taken to correct such immunity defects by appropriate therapy.

During the past 25 years, medical science has made major advances in its knowledge of host defense mechanisms. The host system now includes leukocytes, immunoglobulins, complement and properdin as well as such physical factors as normal skin, mucous membranes and blood vessels. This complex interplay of physiological processes may be altered by disease as well as by medical therapy. Humoral factors, primarily complement and antibody, appear to be the first line of defense behind the mucous membranes and skin. The cells of the body responsible for phagocytosis may be quickly mobilized to contain an infection if they are present in normal numbers. The last line of defense appears to be the cell-mediated functions of the T-lymphocytes and activated macrophages which are required for the killing of intracellular pathogens. Bacterial infections are usually seen when there are defects present in the humoral or phagocytic systems whereas viral, fungal and intracellular bacterial and parasitic disorders are usually seen when cell-mediated immunity is deficient. At the present time it appears that the most common and most important acquired immunological deficiencies found in patients are frequently caused by malnutrition. Infection is often a major factor in precipitating acute nutritional deficiencies.[2] While the interactions of various nutrients are still only partially understood, protein nutrition appears to be very important. Protein and protein-calorie malnutrition are not uncommon. As many as one-half of all patients hospitalized for one week or longer on surgical services are so affected. The immunological abnormalities accompanying malnutrition which predispose to infection are both cellular and humoral. These defects recover promptly on an adequate protein intake. Prospective studies have shown that abnormalities of neutrophil function are the leading immunological defect predisposing to bacterial infection in several types of high-risk surgical and medical patients.[3] Abnormalities of opsonic function have been present in about one-half of the patients studied, and many episodes of bacteremia are preceded by defects of both phagocytic and opsonic function.[4] The clinical evidence for impaired immunity in severe malnutrition consists of a tendency to gram negative septicemia. There is an anergic and afebrile response to infection and the occurrence of gangrene rather than suppuration with tissue infection.[5]

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The medical history and physical examination are extremely important in the evaluation of a patient with an infection for possible defects in the host immune defense system. Particular emphasis must be placed on prior history of infections, either acute or recurrent, family history, drug exposure, exposure to radiation or chemicals and prior medical diagnosis such as diabetes, arthritis or kidney disease. Careful examination for a splenomegaly or lymphadenopathy or for a mild pharyngeal inflammation, subtle rales, perianal tenderness in the absence of swelling or erythema are quite important physical findings. The routine laboratory examinations which the family physician should obtain are serum glucose, blood urea nitrogen, complete white blood count with a differential leukocyte count, and quantitative serum immunoglobulin levels of IgG and IgM. These provide basic data on where to proceed with the diagnostic workup. Tests for B-lymphocyte function can be performed with isohemagglutinin and febrile agglutinin determinations. Also, the Schick intracutaneous test may give valuable information, because up to 50-60% of adolescents and adults have positive tests. T-lymphocyte functions may be evaluated with a PPD, mumps, Candida or trichophytin skin tests as well as by contact sensitization with dinitrochlorbenzene sensitivity. The response of T-lymphocytes should be measured to *in vitro* stimulation by mitogens such as phytohemagglutinin. Complement levels can be measured with total hemolytic complement or C3 levels. If abnormalities in these basic tests occur, investigation should continue with such tests as neutrophil function (ingestion and killing of bacteria), chemotactic response, properdin levels, opsonization, normal complement and alternate complement pathway activation and macrophage stimulation.

Therapy of routine and everyday common diseases frequently affect the patient's ability to combat infection. It has been found that antibiotic therapy is recognized to alter the normal balance of bacteria in the pharynx and gastrointestinal tract. Also, various antibiotics themselves have been shown to decrease the host's immune function and a particular offender of this is tetracycline.[6] Subsequent risk of superinfection by organisms both resistant to the conventional antibiotic and also aided by the antibiotic's suppressive effect on the host immune system is a hazard. Patients under steroid therapy have increased risk of infection. Laboratory studies suggest that steroids decrease mobilization of neutrophils to sites of inflammation, but this has only been found *in vivo* and not *in vitro*.[7] Other defects caused by steroids are decrease in the killing of microbes by both neutrophils and macrophages and interference with the cell-mediated immune response, possibly by alteration of the responsiveness of macrophages to lymphokines. Clinicians who have patients with renal transplants must be aware of their impaired host defense mechanism. The combination of immunosuppressive agents and steroids makes these patients highly susceptible to infections.

Pneumonia in the immunosuppressed patient is quite frequent. Patients receiving cancer chemotherapy or patients with aplastic anemia, defects in humoral factors, or multiple myeloma are extremely susceptible to common bacterial pneumonias. *Mycobacterium tuberculosis* as well as atypical mycobacteria occur in patients with T-lymphocyte defects such as sarcoidosis, Di George's Syndrome or Hodgkins' Disease. Fungal infections are common in patients with T-cell defects and in patients who have a granulocytopenia. Diagnosis often requires aggressive diagnostic procedures such as transtracheal aspiration, bronchoscopy and occasionally transbronchial or open lung biopsy. Bronchoscopic examination in these patients frequently is needed for making the definitive diagnosis, and it will rule out obstructive lesions. X-ray findings are quite helpful. These patients frequently present with fever, shortness of breath and a non-productive cough. A diffuse interstitial pneumonia may be found with infections occurring in children with acute leukemia, primarily in the first six months following initial chemotherapy, or in adults with Hodgkins or non-Hodgkins lymphomas. Two forms of liver disease are particularly important in the host with abnormal defenses. Liver abscesses, particularly abscesses caused by the staphylococcus in a young person, should immediately arouse suspicion of chronic granulomatous disease. Liver scan may demonstrate asymptomatic lucent areas. Frequently the diagnosis of chronic granulomatous disease is made by the failure of the patient's neutrophils to reduce nitroblue tetrazolium dye. Probably a more common form of liver disorder in the compromised patient is hepatitis. The clinical pattern of tender hepatomegaly with atypical lymphocytes in the peripheral blood of a patient with a transplant should raise the physician's suspicion to a cytomegalovirus infection. This also will occur in patients without transplant who are on steroids or cytotrophic drugs for other clinical conditions. Since antibodies to cytomegalovirus are found in 35-80% of the general population, identification of the typical cytoplasmic inclusion bodies in tissue invaded by the virus is required for diagnosis.

The bacterial pathogens, *Mycobacterium tuberculosis* and *Listeria monocytogenes* are commonly associated with the compromised host. These two bacterial pathogens are frequently found in the immunosuppressed patient to be causing a meningitis. The early diagnosis of tuberculosis meningitis requires demonstration of the tubercle bacilli in the CSF sediment. *Listeria* that are frequently present in low numbers and are confused with diphtheroids should be sought carefully. Frequently patients with herpetic infections may present with a diffuse meningoencephalitis, particularly the type 1 Herpes hominis virus. The diagnosis in such patients frequently requires a brain biopsy. Acute bacterial meningitis is frequently associated with a stiff neck and is evaluated expediently by examination of the cerebral spinal fluid. Bacterial meningitis may suggest humoral defects or defects in phagocytic function.

In an effort to restore defects of the host defense mechanism to normal, the physician must give consideration toward the discontinuation of any therapy that may contribute to the abnormality. Hyperalimentation should be instituted in anyone who is unable to take an oral diet for a period greater than five days. Preexisting malnutrition should be corrected with oral supplementation or intravenous hyperalimentation. Deficiencies of the complement components may be corrected in the severely infected patient with the daily administration of cryoextracted plasma, fresh whole blood or cryoextracted blood. The use of packed or washed red cells, albumin or solutions of plasma protein derivative in these patients is discouraged. Transfusion of peripheral neutrophils is of benefit only in severely leukopenic patients. Resistance to infection by selected pathogens may be enhanced by active programs of immunization. Currently programs of immunization for pneumococci are in effect for splenectomized patients. Pseudomonas hyperimmune heptavalent vaccine is currently under intense study in this laboratory for use in burn patients. An area of interest for future therapy is the use of immunoadjuvants to enhance or augment the immune system in the transiently immunocompromised patient. □

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TEACHING HUMAN VALUES TO MEDICAL STUDENTS: SOME PROBLEMS AND IMPLICATIONS

M. OLIVER KEPLER, M.D., M. P. H.*

I taught an elective course to medical students from 1966 to 1972 dealing with the broad area of human values in medicine.[1] I hoped to introduce or reinforce certain concepts such as the nature and importance of the patient's religious beliefs; the place of the clergy in the therapeutic process; the meaning of being a professional person; the historical relationship between medicine and religion; the meaning of medical ethics; some specific ethical issues in medicine; the advocacy role of the physician; the rights of the patient and the social responsibility of the medical profession. I also hoped the course might help produce better physicians by giving them more insight into themselves, their patients and the patient-doctor relationship.

Despite such sanguine hopes, I had no hard proof of their realizations. In former years I had distributed questionnaires to the students who completed the course to determine their feelings as to whether the experience had changed certain of their values.[2] However, such questionnaires are notoriously unreliable. In an effort to test a more objective method, I compared the values of a group of students who *elected* the course with a control group who did not, both before and after the course.[3] In retrospect, I felt that a better comparison would be had between a group of students *selected* to take the course and a control group who did not. Thus, a revised strategy was planned which is the substance of this report.

Despite careful planning, a number of unforeseen difficulties arose in the course of this study which almost certainly affected the validity of the results. However, this study is not reported for its highly valid results or lack of them so much, but rather as a description of one method of securing an objective evaluation of course effects. I further hope that by an honest reporting of the practical problems encountered I might help other investigators minimize such problems. Finally, some speculations will be made on the results insofar as human values courses are concerned.

MATERIALS AND METHODS

Shortly before the fall matriculation, 31 incoming first year medical students at the University of Nebraska were randomly selected from the class roster. These students received a letter from me describing the course in human values and indicating the need for an objective evaluation of the effects of such a course. Their participation in the course was requested and they were told they could refuse without prejudice. Thirty of the 31 students agreed to participate.

Immediately following the fall registration, a battery of "semantic differentials" was given to the entire freshman class. This testing was in no way associated with the project or with the author but administered by a person thought not likely to be identified with the experiment.

Briefly, the semantic differential is a type of scale which measures attitudes or values and has been extensively used.[4] The one we used measured six different dimensions for each of 15 concepts in which we were interested and these items are listed in Tables I and II. (The first concept, Ambulance, was not scored and served only as a buffer item to accustom the students to the mechanics of the test.) Thus, I obtained numerical scores for six different dimensions for each of 14 concepts. Following the completion of the course, the same group of tests was again given to the entire freshman class. An equal number of control students was selected from those who did not take the course, matching them as closely as possible with the experimental group for age, sex, marital status and race. I then had pre and post course scores for both experimental and control groups which were treated as paired differences.

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TABLE I

CONCEPTS

1. Ambulance
2. Chronic Disease
3. Prevention of Disease
4. Humanities Professor
5. Medical Research
6. Patient
7. Surgical Operating Room
8. Total Patient Care
9. Hospital
10. Clinical Medicine
11. Doctor
12. Human Being
13. Medical Educator
14. Patient Rehabilitation
15. Socialized Medicine

The differences, if any, between the experimental and control groups were compared using the "hypothesis of no difference" which is a statistical device for calculating significant differences between the two groups if they truly existed; in other words, the hypothesis was either accepted or rejected. The actual computations were performed on a computer using the program known as the Statistical Package for the Social Sciences or SPSS.

The class with which we are concerned here entered medical school in the fall of 1971 and was generally characterized by students who were outspoken and sometimes rebellious; a phenomenon that was noted in many medical schools at that time. Of the original group of 30 students randomly selected to take the human values course, only seven honored their signed pledges. These defections meant that the students could no longer be considered as randomly selected and this fact plus the reduced numbers would adversely affect the validity of the results. However, for reasons stated earlier, I decided to complete the study as planned.

TABLE II

DIMENSIONS

A. Evaluation
B. Potency
C. Activity
D. Tautness
E. Novelty
F. Receptivity

RESULTS

The final scores are listed as probabilities in Table III. The first concept, Ambulance, was not scored for reasons stated earlier. The figures in Table III represent the probability that there is no difference between the experimental and control groups for any given dimension. Thus, the first dimension, *evaluation*, for the concept of Chronic Disease is .95 meaning that there is a 95% probability that there is no difference between the two groups for this dimension. A total of six dimensions for each of 14 concepts was computed for a total of 84 probability figures. Only the numbers in parentheses indicate a significant probability of difference between the two groups for these dimensions; a total of 12 in all. These particular figures indicate a probability of 5% or less that there is no difference between the two groups for that given dimension, or stated another way, a 95% or greater probability that there was a difference between them.

Six concepts showed no difference in any dimension, four concepts showed a difference in one dimension and four concepts differed in two dimensions. It is notable that all of these 12 dimensions showing significant differences were negative for both groups, that is, both groups gave a lesser response to these dimensions with the passage of time. Furthermore, the experimental or course-taking group differences were more negative than those of the control group.

The question then arose: Was the experimental group more negative than the control group before taking the course? Using another statistical procedure, the pre course test scores for the

DIMENSIONS

CONCEPTS	A. Evaluation	B. Potency	C. Activity	D. Tautness	E. Novelty	F. Receptivity
1. Ambulance	----	----	----	----	----	----
2. Chronic Disease	.95	.89	.41	.56	.21	.17
3. Prevention of Disease	(.01)	.50	.39	.65	.85	.11
4. Humanities Professor	.97	.56	.94	.37	.20	.80
5. Medical Research	(.03)	(.04)	.63	.20	.36	.60
6. Patient	.90	.70	1.00	.73	.78	1.00
7. Surgical Operating Room	(.03)	.15	(.02)	.66	.16	.11
8. Total Patient Care	(.02)	.31	(.04)	.66	.21	.23
9. Hospital	.39	.08	.08	.93	.89	1.00
10. Clinical Medicine	.35	.61	.34	.41	.22	1.00
11. Doctor	.20	.10	.84	.59	.28	.27
12. Human Being	.46	.82	(.03)	.66	.72	.24
13. Medical Educator	.36	(.04)	.60	.57	1.00	.21
14. Patient Rehabilitation	(.01)	.75	.80	.75	.46	(.02)
15. Socialized Medicine	.31	.23	.93	.18	(.05)	.82

Quantities in parentheses are below the significance level α = .05. In these instances the hypothesis of no difference between the two groups is rejected.

TABLE 3

PROBABILITY THAT EACH TEST STATISTIC WILL
FOLLOW THE STUDENT T-DISTRIBUTION GIVEN
BY THE HYPOTHESIS OF NO DIFFERENCE IN THE
COMPARED GROUP MEANS

two groups were compared on three of the more important dimensions for all 14 concepts.** Actually, the experimental group was somewhat more positive than the controls in five of the dimensions compared although the differences were not marked. Apparently the two groups started out with reasonably equal values for the dimensions tested.

DISCUSSION

Disappointingly, students who were selected to take the Human Values course showed none of the benefits I had hoped to see. It cannot be fairly stated that either the course or the selection process made the selectees more negative than the controls but it can be fairly stated that the 30 students originally selected were rebellious and not easily held to their pledges. To have only seven students remaining from an original group of 30 selected to take the course does ruin the random selection process and probably results in a biased sample of medical students.

It is not possible to do more than speculate on the observed differences in the two groups. Looking at the results which showed significant differences, one sees that the experimental group gave a lessened *evaluation* for the Prevention of Disease concept. The course did emphasize prevention of disease as the first goal of medicine so this result was surprising; however, others have noted similar attitudes toward preventive medicine among medical students. The lessened *evaluation* and *potency* values given to the Medical Research concept are not surprising in view of the students' increasing contact with research programs. The results for Surgical Operating Room were expected and this particular concept was not judged important. However, the lessened ratings for Total Patient Care were disappointing since this is judged an important concept. One possible explanation is that although course students were involved in discussions about the desirability of total patient care, they seldom saw this concept practiced. This same theory might explain negativity toward Prevention of Disease; again, this is something that is not widely practiced. Other researchers have noted similar negative effects in medical students.[5] The lessened ratings for the concepts of Human Being and Medical Educator were not unexpected and were relatively unimportant in terms of course objectives. Furthermore, the lessened *novelty* aspect of Socialized Medicine might be regarded as a normal outcome. If we might assume that these lesser ratings given by the experimental group have some validity, why should this be so? For some possible answers, we must speculate a little for there are not a lot of hard data in this field.

There is, however, a large number of studies to show that medical students become increasingly cynical and disenchanted toward the medical education process itself; for example, the sheer overload of learning. Also, it is becoming more apparent that "enrichment" courses like the one I offered in human values might accentuate student negativism and cynicism for reasons hinted at earlier. There are many students who perceive that medical education doesn't always practice what it preaches and this can certainly accentuate cynicism. Thus, in medical school we hear the virtues of preventive medicine extolled but do not see it widely practiced. We hear that primary care is essential and prestigious but we see that research and super-specialization is more emphasized than primary care. Ambulatory clinics lack the glamor of the operating room and the "LMD" is still a pretty inept person in the grand rounds protocol. We hear that compassion and concern for patients are necessary but as students we fail to see much compassion and concern accorded us. We hear that medical students should be broadly educated in the humanities and behavioral sciences as well as medical science but it is medical science subjects and it is high grades in these subjects that get most of us into medical school. We hear that health education is important but we rarely encounter a patient who has had his condition thoroughly explained to him so that he can understand it and effectively collaborate in the treatment process. There are many other examples one might cite.

The wonder of it all is that so many physicians do become caring and concerned practitioners and eventually lose most of their earlier cynicism. What I am saying is that individual courses in human values, ethics, preventive medicine, etc. are often perceived as "non-medical" by the student and their content will be largely ignored when compared with the general milieu of medical education. Hence, the course takers in this

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** The dimensions which were compared were *evaluation*, *potency* and *receptivity*. ($p < .10$)

study may well have had a faster erosion of values than the control group because the course effort was seen as hypocrisy. The obvious answer is to work for a consistent atmosphere in medical education which does practice that which it says is good.

Should such efforts as a human values course be abandoned? I'm not ready to say that. An earlier study which I cited in the introduction compared a group of students who *elected* this course with a control group who did not. In this case, the course takers were more resistant to erosion of values than the controls. This might mean that those who chose such a course had more positive attitudes toward the values we were stressing. Taking another leap, it is possible that applicants selected for admission to medical school might be chosen on the basis of their values as well as their grade point averages.[6] I am inclined to think that until this happens, enrichment courses can strengthen and reinforce preexisting values held by certain students and I trust that this could make them better practitioners of the art. Much more research is needed in this entire area and happily there is increasing interest in this subject.

SUMMARY

To determine whether the taking of a course in human values would change certain values held by medical students, a controlled study was performed. Student defection and other problems are discussed. In this limited and imperfect study there was no appreciable impact of the course on the values which were tested. Both experimental and control groups showed erosion of values during the first year. It is possible that courses of this type may alienate certain students and increase their cynicism but it is equally possible that other students will find it helpful in reinforcing certain previously held values. Some implications for medical education are raised. □

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Editorials

TO CARE AND NOT TO CARE

"The problem with medicine nowadays," opined my prosperous businessman cousin as he leaned back from the table after savoring Thanksgiving dinner, "is that doctors are too scientific and don't seem to *care* about their patients and take the *time* to talk to them." Meekly, I argued that there are only so many hours in the day. Given the choice between the best scientific medicine and caring talk, I'd prefer to pay *my* doctor for the former; my health matters more to me than his sympathy. I went on to elaborate about some of the complexities of the doctor's many dilemmas — knowing all the time that my cousin would insist upon the last word. He did.

In this issue of *The Journal*, Dr. Oliver Kepler discusses some of the problems of teaching human values to medical students. He met many frustrations in teaching an elective course, which had been designed to provide the students "more insight into themselves, their patients and the patient-doctor relationship." "The wonder of it all," Dr. Kepler tells us, "is that so many physicians do become caring and concerned practitioners and eventually lose most of their earlier cynicism."

Each of the points made by my cousin, and most of the issues raised by Dr. Kepler, were raised 51 years ago by Dr. Francis Peabody in his eloquent commencement address at Harvard Medical School. Peabody acknowledged the criticisms that medicine had become "too scientific" at the expense of compassion, that approximately one-half of patients who present themselves to physicians' offices bring with them symptoms lacking a diagnosable organic basis, that medical training provides little foundation for dealing with many of the situational problems to be encountered, and that the treatment of a disease process differs from the management of a patient. His final statement lingers on: "One of the essential qualities of the clinician is interest in humanity, for the secret of the care of the patient is in caring for the patient."[1]

Teachers with Peabody's insight and eloquence are rare. One such teacher, Dr. Philip Tumulty of The Johns Hopkins University School of Medicine, retired this year and, like Peabody, gave a commencement address which may linger on to inspire future generations. Beginning with the simple fact that the patient is, first of all, a person, Dr. Tumulty elaborates that the patient "is a very special kind of a person because he is a sick person." Few professors have surpassed Tumulty's ability to enumerate the factors which contribute to effective patient management. Pertinent to *caring* in our era, this reminder by Dr. Tumulty stands out: "Such terms as primary care, secondary care or tertiary care are, in fact, meaningless. There is really a choice between only two kinds of care: good or bad, adequate or inadequate, complete or incomplete."[2]

Can caring be taught? Can a course aimed at conveying the principles of caring compete for the student's interest with the rigorous, traditional scientific subject matter? Is it better to defer the lessons in caring to some later date in the physician's education — perhaps to the residency years, or perhaps to the trial and error of practice?

A great deal has been written about these issues. The acceleration of technology and of medical knowledge have made Dr. Peabody's famous passage seem simplistic. The decisions which the physician faces, confronted with a sick patient, are often extremely intricate. The thoughtful physician might, for instance, *care* considerably about whether to have his patient undergo computerized tomography as part of a headache workup—will the patient actually *appreciate* the complexity of his *caring* decision that the patient's (or his insurance carrier's) money might be invested more wisely, since the headache is much more likely to represent tension than meningioma? Probably not.

Recently, a medical student *pleaded* with us for more scientific information about caring as part of the medical curriculum.[3] Arguing that "the notion that caring comes naturally to the physician in training is a fantasy," he went on to tell us:

> "Caring . . . (now) carries different connotations and responsibilities from those of 50 years ago when Peabody wrote about it. One can no longer consider the caring role of the physician without pondering issues such as how medical care is delivered to the poor, or the wise use of increasingly expensive technology. Caring certainly includes the noble feelings expressed by Peabody, but it also encompasses the hard realities of equity and cost. A real danger in failing to provide education for caring is that too few physicians are trained to grapple with these latter issues."[3]

This author and others[4] agree that the notion of caring encompasses a number of aspects of human behavior which should be amenable to scientific study.

It is somewhat surprising that few of the essayists who have written about caring in medical practice seem to have defined their word. The usual synonyms for "caring" would be "concerned" and "interested." A practical problem for the physician — given the limitations of his time — is that he must both *be* concerned and *convey* this concern to the patient. Let us assume that a physician has but ten minutes to spend with a sick person before he must hurry to the next emergency. Is it preferable to spend that time obtaining a crucial bit of scientific information — for instance, examining the peripheral blood smear, urine sediment, or sputum gram strain — or should he spend that time reassuring the patient and his relatives that everything possible is being done? If he chooses the former course, he has probably done what is best for the patient's health, but risks losing the patient's confidence. If he chooses the latter, he gains the patient's confidence for the moment, but knows that he has, in fact, not done his best work. The physician is in a classic double-bind situation. Nearly always, the basic cause is the limitations of his time.

Caring takes time. As Dr. Tumulty states, "Time personally spent with the patient is the most essential ingredient of excellence in clinical practice. There are simply no short cuts and no substitutions." A sad commentary of our era is that "time for caring" is seldom if ever recognized by third party payors as an allowable charge. As a practicing internist put it, "A system of physician compensation has developed in this country so that the less a doctor talks to you and the more he does to you the more he is paid."[5] One should not assume that the technical doers do not care; they may care a great deal, and their pride in technical excellence is certainly in the patient's best interest. One should not even assume that physicians who seldom if ever talk to patients do not care — I think especially of a famous surgical pathologist who rarely saw patients, but could remember the names of the persons associated with his slides and bits of tissue years later. But we should insist that the considerable *time* needed for caring and conveying our sense of caring deserves some reimbursement (my businessman cousin was quick, after all, to place a monetary value on *his* time).

Finally, the notion of how to care *appropriately* deserves attention in the medical curriculum. How, for instance, should the student be taught to care for the patient with literally bottomless needs for human sympathy and compassion? Such patients, familiar to all physicians' offices, have the potential to drain their doctors' emotional and physical resources to the point that appropriate care for all of their patients becomes compromised. The professional person's caring demands some degree of detached objectivity.

Yes, Peabody's famous passage is too simplistic. Caring must be learned, and hopefully can be taught. Its limits require better definition. As T. S. Eliot put it in "Ash-Wednesday": "Teach us to care and not to care. . . ."

— CSB

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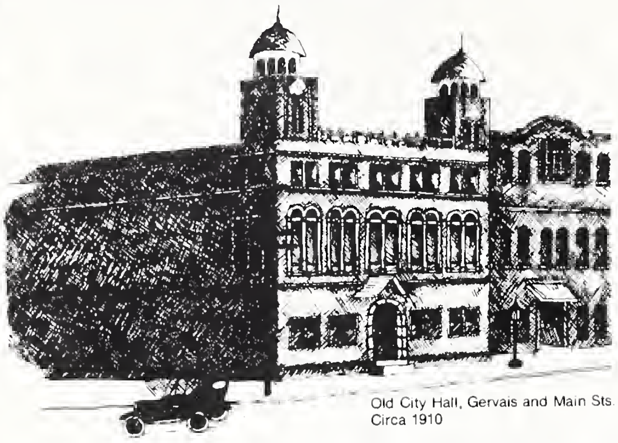
TAX SHELTERS?



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NATIONAL HEALTH INSURANCE

Editor's Note: The writer is an assistant professor of Economics at Clemson University. This essay, reprinted with permission, first appeared in the Messenger *of Clemson, S. C.*

Sen. Edward Kennedy, speaking recently to the AFL-CIO, said the people of this nation need national health insurance. Furthermore, he said that Proposition 13 proved that we, the people, want the federal government to take care of our health needs because medical costs rose by $7 billion last year. It is necessary to examine these statements.

Do Americans need to be supplied with health services from the same people who brought us Amtrak and the post office? At present 90 percent of the population is covered under some sort of medical insurance. Furthermore, out of the remaining 10 percent a good number are covered under welfare, medicaid and other government programs. Hence, Kennedy is willing to impose costs on more than 90 percent of the population to help less than 10 percent.

How high will these costs be? One estimate made in the Sept. 4 issue of *Business Week* is that national health insurance programs as proposed by Kennedy will cost you, the taxpayer, $225 billion the first year of operation, and considerably more thereafter. That is such a large number it must be broken down.

The cost to an average family of four would be $4,091 more a year or $340.90 more a month in taxes. In effect, the program would increase the size of the federal government by 50 percent in the first year that the new legislation is in effect.

Wasn't the idea behind Proposition 13 that taxes would be lowered and that the size of the government would become smaller? If so, then how is increasing the average tax burden $341 a month for the average family in accordance with the sentiment for less government and lower taxes?

As with most of these income redistribution schemes, income will be redistributed from the middle class to certain "privileged" (as determined by HEW rules) classes. In this case we would expect that the medical services that the middle class now receives will be greatly reduced.

Prices allocate resources. Not all prices are expressed in dollars; some are in time. When medical services are desired, you pay both dollars and time. Waiting in the office for the doctor can be very expensive in terms of time if you have to miss work.

With national health insurance the limited amount of medical services will be allocated only by time. For many middle class individuals, hours spent waiting in the doctor's office will not be worth the medical services received for minor ailments or preventive medicine. For lower income people, the cost of waiting is less. In effect, then, since the national health insurance program is not designed to increase the overall amount of medical services available, it will take medicine away from the middle class and redistribute it to the lower income class.

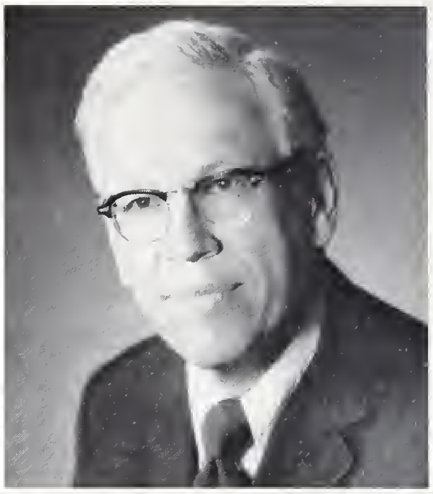
There is also a question of efficiency which must be considered. In almost all cases, nationalization has resulted in reduced efficiency. Amtrak is a good example. The inefficiency is due to a change in the incentive system. Under a private enterprise system, people are rewarded for innovation and diligence and punished for failure. Under collectivism people are punished for innovation (don't rock the boat) and often not punished for failure.

We all know how hard it is to fire a federal employee. Now imagine what happens to efficiency when national health insurance is put into effect. Imagine being a patient lying in a hospital bed getting medicine from nurses who do not get punished if they give you the wrong medication — or for that matter ignore you altogether.

In short, what we stand to gain from Kennedy's proposal is more government but less medical care. The appeal to Kennedy is understandable. But for the average U. S. citizen, careful consideration of the program may only make him sick.

JOSEPH PRINZINGER
Clemson University

President's Pages



AMA AND NHI: A WELCOME CHANGE IN THE HOUSE OF DELEGATES

There's an old saying: "You win some, and you lose some." For years, when returning from AMA meetings, I have wondered about the "winning" part. Chicago — December, 1978 — was different. I came back exhilarated instead of depressed. For the first time in eight years, the AMA House of Delegates has finally refused to approve sponsorship (and introduction into the 96th Congress) of a Comprehensive (National) Health Insurance Bill, this time re-named "The Health Insurance Improvement Act of 1979."

A remarkable series of events occurred, and a lot of jigsaw pieces had to fit into place before it was over. Undoubtedly, the major credit goes to Dr. Joseph Von Thron, Delegate from Florida, who delivered the most effective speech I have ever heard on the floor of the House of Delegates. In deliberately measured phrases, and low tones — with Delegates leaning to catch his every word — he built up the case *against* passing Board of Trustees' Report MM, which had been recommended by Reference Committee B.

Then he moved for passage, instead, of Resolution 62, submitted by the Florida Delegation. This Resolution stated:

> "*WHEREAS, it is a recognized fact that Americans have the best medical care delivery system in the world today, and most polls indicate the public is generally satisfied with both the quality and quantity of medical service; and*
> *WHEREAS, the polls have consistently reflected that the American people hold their individual physician in higher regard and trust than any other professional; and*
> *WHEREAS, it is this very personal doctor-patient relationship that will be dismantled and ultimately destroyed by National Health Insurance; and*
> *WHEREAS, the primary thrust for a nationalized, socialized system of medical care has continuously come from the political arena where logic is often lacking; and*
> *WHEREAS, at this time it would be unwise for organized medicine, at any level, to sponsor or support federal legislation to further nationalize or socialize physicians' services in this country; and*
> *WHEREAS, there is a need to improve our present system – not by discarding or disregarding our present one; therefore, be it*

RESOLVED, that the American Medical Association recommends to the Congress of the United States of America modifications to our present health care system embodying the following principles:

1. *Requiring minimum standards of adequate benefits in all health insurance policies sold in the United States with appropriate deductible and co-insurance.*
2. *A simple system of uniform benefits provided by the federal, state, and local governments for those individuals who are unfortunate enough (through no fault of their own, i.e., age, disability, financial hardships, etc.) not to be able to provide for their own medical care.*
3. *A nationwide program by the private insurance industry of America (and government, if necessary for reinsurance) to make available catastrophic insurance, for those illnesses and individuals where the economic impact of a catastrophic illness could be tragic. All catastrophic coverage should have an appropriate deductible and co-insurance to make it economically feasible and to avoid abuse. A program developed pursuant to these principles should be administered at the state level with national standardization through federal guidelines.*

Much maneuvering then ensued. Forceful arguments were heard on both sides of the question. A motion to shut off debate was passed by the required two-thirds while many, including AMA President Thomas B. Nesbitt, were still waiting at the microphones. Finally, the Florida Resolution was passed by a stunning two to one margin, and was later referred to the Board of Trustees for implementation.

This action was not as clear-cut as it would have been if the House had passed our own South Carolina Resolution: " . . . that the American Medical Association will not introduce any National Health Insurance proposal in the 96th Congress, and will oppose any proposal for National Health Insurance which requires substantial additional governmental expenditure," but it was definite enough. It signalled a profound change in the mood of the House and a firm determination not to endorse the "Health Insurance Improvement Act of 1979." A draft of this Bill had been handed to the Delegates for the first time at 4:30 p.m. on the opening day of the meeting, despite instructions to the Board of Trustees at the 1978 Annual Convention that any AMA-sponsored Bill should be circulated to the members as early as possible before the December meeting, "so it may be studied in detail before that session."

There were plenty of other important topics, of course, and not all of them were pleasant in their solutions. Special legal counsel, Mr. Newton M. Minow, gave a detailed "Report on Litigation," which included discussion of the chiropractic suits against the AMA, and the recent ruling against the AMA by a Federal Trade Commission judge with regard to our Principles of Ethics. These were sobering issues, and had to be acted upon after thoughtful deliberation — for the most part upholding the actions taken by the Board of Trustees.

But they are another story, which you will read about elsewhere or in later issues of this *Journal.* Clearly, the memorable event of this session was the decisive vote *not to introduce* the proposed "Health Insurance Improvement Act of 1979."

JOHN C. HAWK, JR., M.D.
President

INFORMATION FOR AUTHORS

We encourage original articles and letters to the editor of potential benefit and interest to the members of the South Carolina Medical Association.

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PRIORITY FOR PUBLICATION: We give priority to the following categories: (1) concise review articles of clinical topics; (2) case reports and other original observations by practicing physicians; (3) articles devoted to social, economic, or ethical topics; and (4) information from government and other agencies pertinent to the health care of South Carolinians.

Of lower priority, but still welcome for consideration, are articles in the following categories: (1) historical or philosophical essays, and (2) original scientific investigations of a specialized nature.

REVIEWING AND RESPONSIBILITY TO READERSHIP: We will make every effort to review manuscripts promptly. All manuscripts will be reviewed by our editorial office, and when indicated the opinions of outside consultants will be solicited.

We welcome criticisms of journal content by members of the South Carolina Medical Association.

REPRINTS: These will be made available by the publisher at established rates, at the time of mailing of galley proofs.

LENGTH OF ARTICLES: We prefer concise articles of approximately 2500 words (approximately 8 typewritten pages, double-spaced), with no more than ten references.

We regret that space considerations limit our ability to publish longer articles, and request that authors adhere to the above guidelines. Similarly, tables and illustrations (see below), should be kept to a minimum, and be specific and pertinent.

MANUSCRIPTS: These should be typewritten, double-spaced, and on one side of the paper. The original and one copy should be submitted. The title page should indicate the title, author(s), author's address, and academic appointments, if any. We request that the author's name not appear on subsequent pages, to permit "blind" review of the article, when desired. Authors should retain one copy for use in proofing.

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REFERENCES: These should be cited consecutively in the text, in superscript, e.g., "Kaunitz and Lindenbaum^3 . . ." We recommend no more than ten references, selected from more recent publications in accessible journals in most instances. When an extensive bibliography is desired, this can be accomplished at the author's expense or by noting that "additional references are available from the author upon request." Standard journal abbreviations should be used, with the style for journal articles being as follows:

3. Kaunitz JD, Lindenbaum J: The bioavailability of folic acid added to wine. Ann Intern Med 87: 542-545, 1977.

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VOLUME 75 FEBRUARY 1979 NUMBER 2

THE BALLOON CATHETER TECHNIQUE IN COLOSTOMY REVISION

JOSEPH HODGE, M.D.*

Complications subsequent to colostomy are retraction, stenosis, herniation, hemorrhage, perforation, gaseous distention, skin irritation, gangrene, abcess and prolapse of the intestine.[1-3]

The purpose of this paper is to present the balloon catheter technique as an aid in revising the end colostomy stoma incident to stenosis and retraction following Miles resection for carcinoma of the rectum.

REPORT OF CASE

This 64 year old female was admitted to the Spartanburg General Hospital August 21, 1977, complaining of difficulty in having bowel movements through her colostomy site. She experienced colicky right and left lower quadrant abdominal pain upon irrigating the colostomy and passed blood. In February 1972, patient was found to have an adenocarcinoma of the rectum. On March 6, 1972, she had a combined abdominal perineal resection for adenocarcinoma of the rectum which penetrated the muscularis but no involvement of the adjacent lymph nodes. In December 1972, barium enema and bone survey disclosed no evidence of recurrent or metastatic disease. She was asymptomatic for five years. On follow up visits no abdominal masses nor evidence of metastasis to liver and lymph nodes were noted.

In May 1977, the patient developed psychiatric problems and during her confinement in the psychiatric ward she neglected to care for her colostomy and gained 50 lbs. of weight while on Etrafon, Trilafon and Artane.

The pertinent physical findings during the August 1977 admission were stenosis of the colostomy site with retraction, granulation tissue and perforation at the skin level. It was difficult to insert the little finger into the stoma. No abdominal masses, hepatomegaly nor evidence of hepatic and lymph node metastasis was noted.

Chest x-ray, liver scan, bone scan were normal. Barium enema was performed by introducing a 14 French Foley catheter and there was no evidence of ulceration nor malignant changes. On August 25, 1977, patient had revision of colostomy stoma utilizing the balloon catheter technique. The post-operative course was uneventful. Her last follow up visit on May 22, 1978, revealed her colostomy to be well functioning with no further evidence of stenosis or retraction. (Fig. 4)

OPERATIVE TECHNIQUE

Following the usual prep and drape and under satisfactory general anesthesia and following walling off of the operative site with Steri-Drape, a balloon catheter #14 French, was inserted into the colostomy opening at the base of the skin and advanced forward. The 30cc Foley catheter balloon was inflated (Fig. 1) and withdrawn, thereby occluding the stoma and preventing fecal contents from contaminating the operative field. With retrograde traction on the catheter by the assistant (Fig. 2), the walls of the stoma are always palpable. An incision was made circumferentially around the skin and colostomy stoma until the base of the colostomy at the fascial level was reached. By palpating the serosal walls of the colostomy, the proper cleavage plane was easily developed down to the external oblique aponeurosis and all redundant tissue and stoma mucosa were excised without injury to the colon. The oozing edges of the stoma were approximated with a 3-0 plain catgut. The stomal mucosa was everted and anchored to the skin level with 4-0 interrupted silk sutures. The wound, superior and inferior to the stoma, was approximated with interrupted appropriate catgut sutures and the skin with 4-0 interrupted silk sutures.

---

* 856 North Church Street, Spartanburg, S. C. 29303

After completion of the revision and repair of the abdominal wound, the wound was dressed, the balloon deflated and removed. (Fig. 3) The stoma should admit two fingers. The stoma was packed with Idoform gauze for approximately 12-24 hours. (Fig. 3)

DISCUSSION

Stricture can occur at the skin level when the stoma retracts or secondary to infection incident to trauma or perforation of the stoma with the irrigating catheter. This complication can be prevented by adequate immobilization of sufficient bowel above the skin, thereby preventing infection at the site of the stoma. Strictures at the fascial level may be treated by excision of an adequate surrounding fascia. When the mucosa of the stoma is sutured to the skin, primary healing occurs thereby minimizing subcutaneous infection and scarring.

Retraction may follow the attempted passage of a hard stool. Adequate mobilization and fixation of the colostomy stoma to the lateral wall with interrupted sutures through the cut edges of the mesentary and appendages will also prevent this complication.

The patient reported had a good function colostomy for 5 years following surgery, but because of psychiatric problems, she gained considerable weight, neglected the proper care and irrigation of her colostomy. As she attempted to irrigate the colostomy, perforation occurred at the skin level and with the passage of hard stools the cycle for infection, stenosis and retraction of the stoma occurred.

SUMMARY

The balloon catheter, when inflated, serves as an anatomical guide in revision of colostomies thereby preventing entry and injury to the bowel wall. With retrograde traction the balloon occludes the stoma site thus preventing contamination of the operative field with fecal contents. This technique efficaciously facilitates stoma revision. ☐

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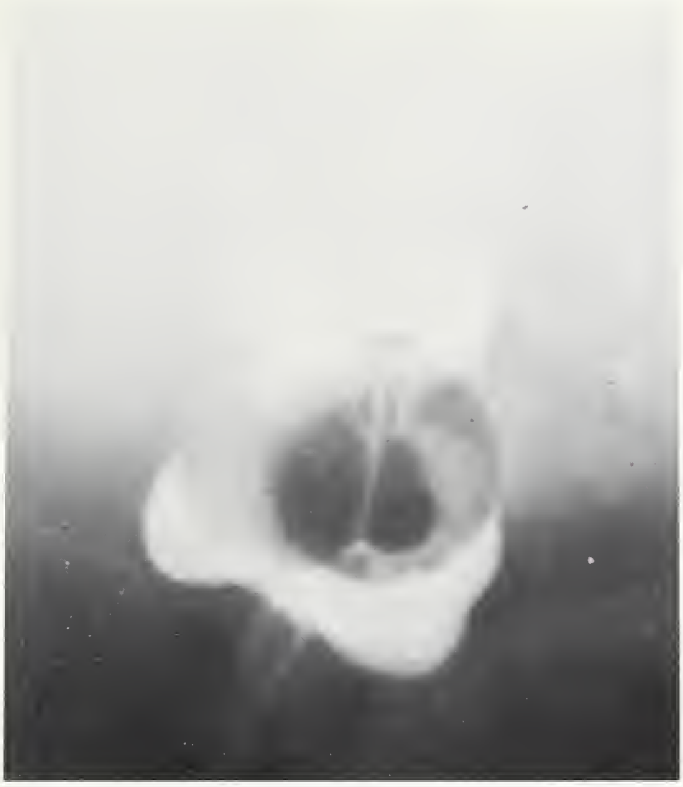

FIGURE 1 — Introduction of the Foley Catheter with inflation of the balloon.

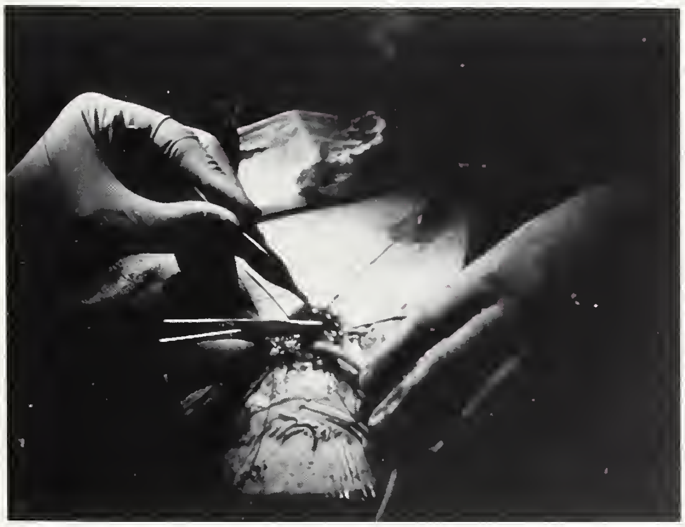

FIGURE 2 — Retrograde traction on the catheter by the assistant.

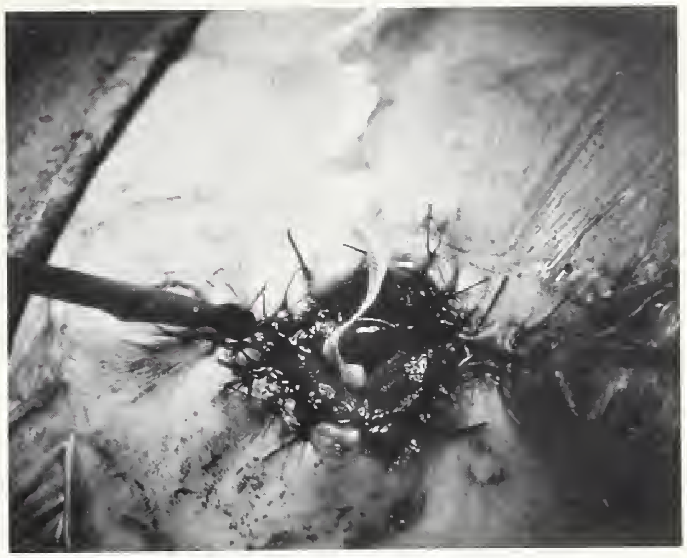

FIGURE 3 — Balloon deflated and removed.

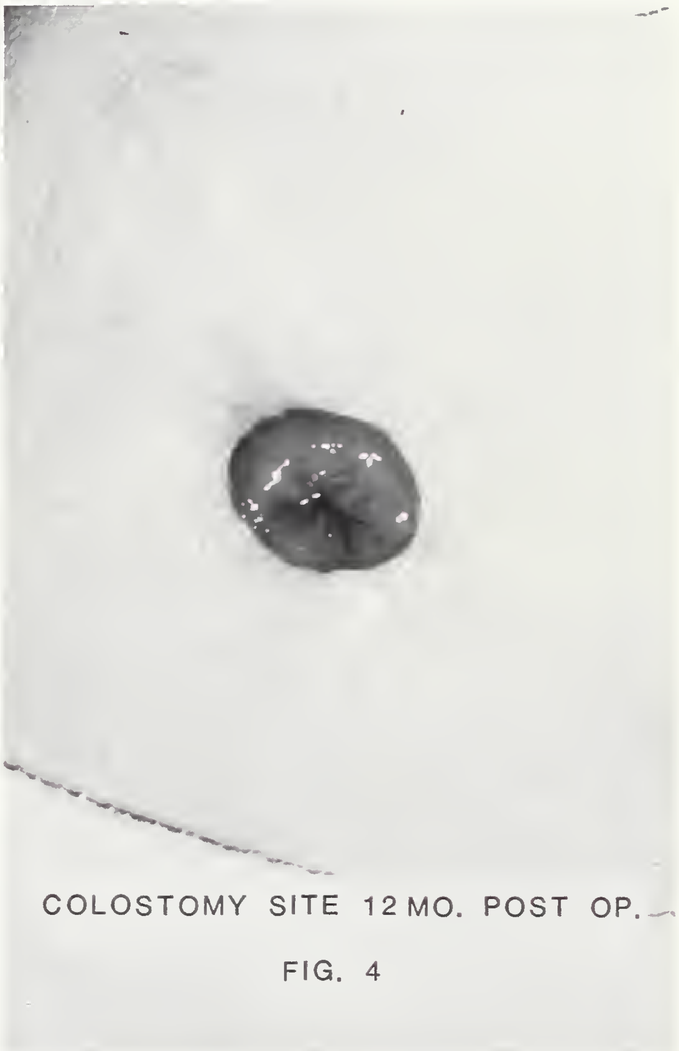

FIGURE 4 — Colostomy stoma 12 months post-op.

URODYNAMICS: A NEW FIELD IN UROLOGY

FREDERICK J. GOULDING, M.D.*

Although the process of micturition was studied by physiologists as early as 1778,[1] urodynamics is a relatively new field in urology that deals with the measurement of flow, pressure, and electrical muscle activity associated with the storage and passage of urine. Measurement of these parameters has been going on for some time, but recently the ability to measure and record all of them accurately and simultaneously has been made possible by compact and widely available recording units (Fig. 1). The following case reports are meant to serve as examples of the usefulness of urodynamics. A brief summary of each of the tests which can be used singly or in combination to diagnose and evaluate the disease state and its treatment follows the case reports.

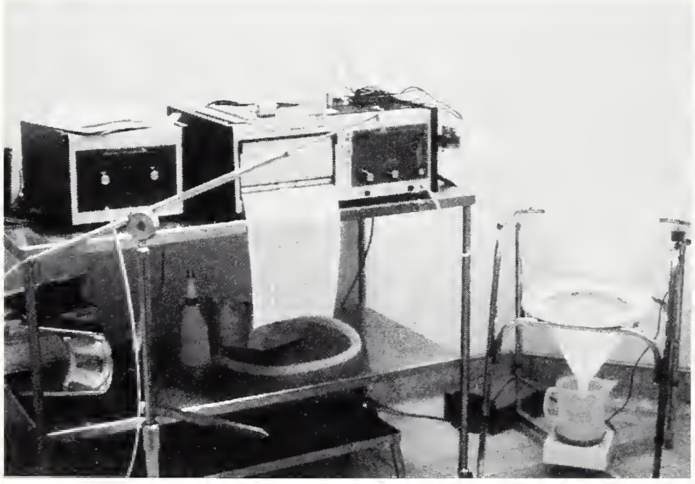

FIGURE 1: Four channel urodynamic equipment used at North Trident Regional Hospital for the last 2 years.

CASE REPORTS

CASE 1: A 37 year old male underwent a C_{6-7} spinal cord trauma 12 years ago with resultant quadraplegia of a C_6 level. He was initially managed with TUR-prostate and bladder training and developed a reflex bladder requiring only an exdwelling catheter. His post void residuals remained low, his urine sterile on antibiotics, and his upper tracts stable on serial IVPs (Fig. 2) until the onset of a *Pseudomonas* and *Klebsiella* urinary infection which was adequately treated. Following the treatment his IVP showed bilateral ureterohydronephrosis which persisted despite sterile urine, and post void residuals increased to 300-400 cc (Fig. 3). Urodynamic testing consisting of simultaneous sphincter EMG activity, flow rate, and intravesical pressure demonstrated bladder-sphincter dyssnergia. Various pharmacologic agents were tried but without success, and the patient underwent an external urinary sphincterotomy with improvement of his upper tracts and resolution of the large post void residuals (Fig. 4).

CASE 2: A 41 year old male who had been a C_5 quadraplegic secondary to trauma for 19 years and had undergone bladder training developed a reflex bladder and wore an exdwelling catheter. He underwent a TUR-prostate 5 years prior to this admission for outlet obstruction. He developed a series of urinary tract infections which culminated in acute pyelonephritis with sepsis. His upper tracts remained stable on IVP but post-void residuals of 500-600 cc prevented clearing of the bacteriuria. Urodynamic testing revealed bladder-sphincter dyssnergia and pharmacologic manipulation was unsuccessful. An external sphincterotomy was performed with the resoltuion of the large residuals and infection.

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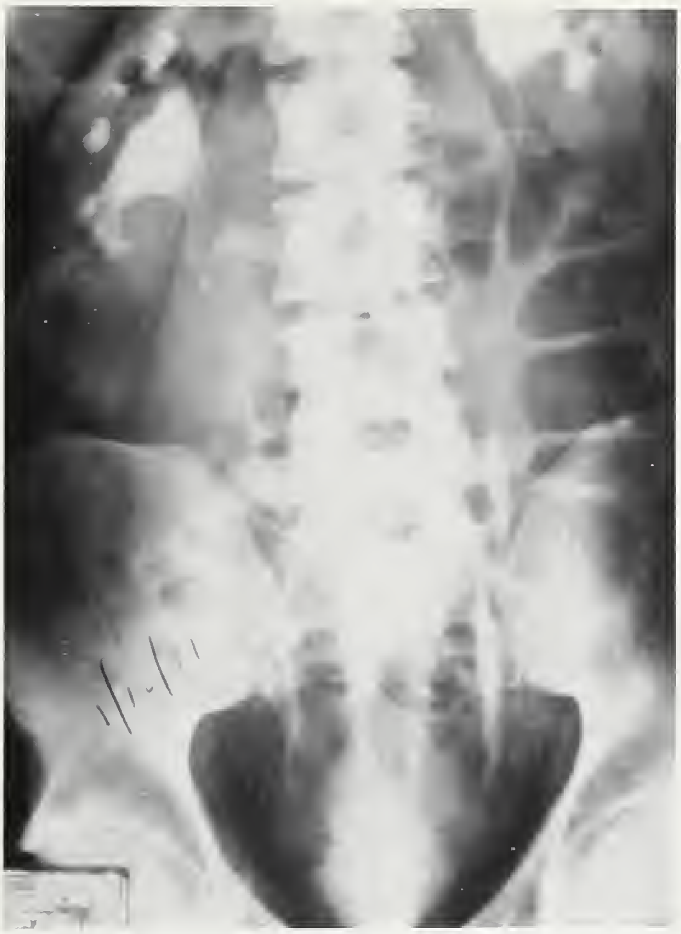

FIGURE 2

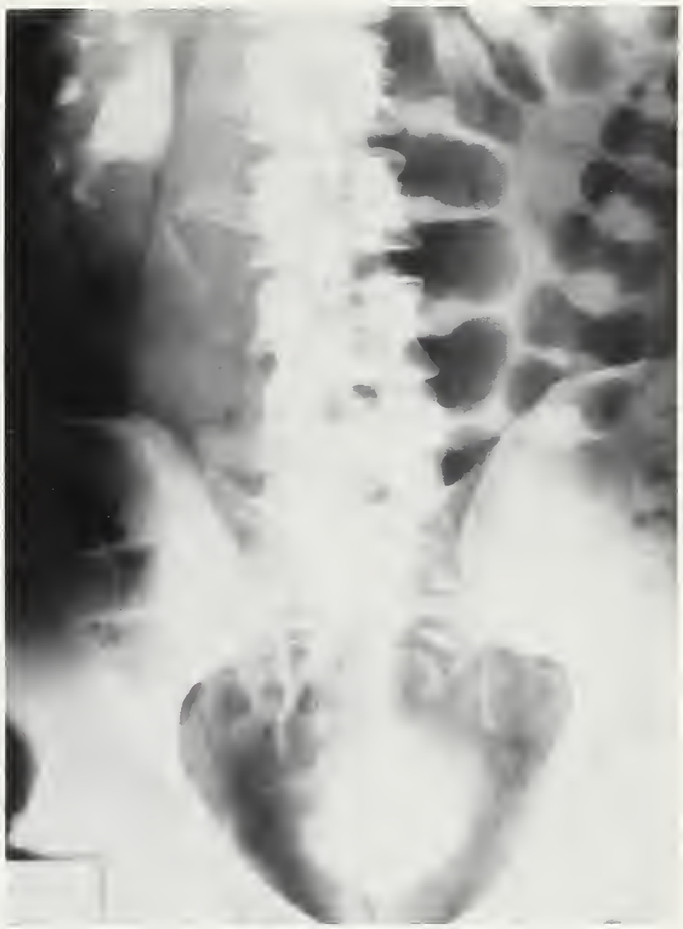

FIGURE 4

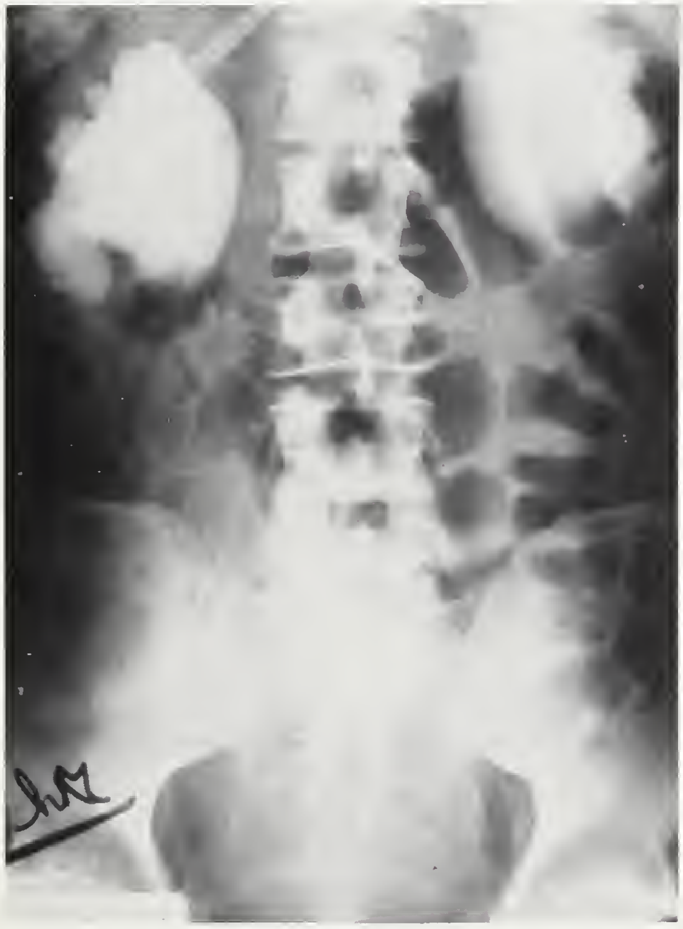

FIGURE 3

CYSTOMETROGRAM (CMG)

Cystometry prior to the past decade was performed with water infusion but recently the use of CO_2 with rapid infusion rates has allowed the subject to be quickly and repeatedly evaluated in the sitting, standing, walking positions,[2] and with medications. The principle observations to be made are: (1) the presence of a detrusor reflex and the patient's volitional control over this reflex or absence of this detrusor reflex (areflexia) even with detrusor activation procedures such as change in posture, administration of bethanechol or rectal or urethral distention; (2) Proprioceptive sensation as tested by first sensation to void or discomfort with fullness and exteroceptive sensation as measured with sensation to hot or cold water; and (3) Tonus limb slope, the elastic property of the detrusor tissue to filling.

Detrusor hyperreflexia (reflex contraction that the patient cannot voluntarily suppress) is frequently seen in patients with spinal cord trauma, stroke, Parkinsonism, multiple sclerosis, amyotrophic lateral sclerosis, arachnoiditis, and dementia. Areflexia is often seen in a central herniated disc, diabetes mellitus, spinal cord birth defects, and cauda equina and conus medullaris injury. Increase in the slope of the tonus limb is noted in patients with increased collagen content secondary to chronic bladder infection or prolonged drainage; flattening of the tonus limb results from over distention and permanent alteration in the elastic characteristics of smooth muscle and collagen.[3] Interpretation must be made in conjunction with the history, urologic and neurologic exam.

SPHINCTER ELECTROMYOGRAPHY

Electromyography may be of the external urethral sphincter, pelvic floor musculature, or anal sphincter and may be performed by use of needle electrodes, catheter electrodes or an anal plug. For most patients, the pattern of activity in the periurethral striated muscle during bladder distention and contraction is similar to that in the anal sphincter except in those patients with stress incontinence, periurethral trauma or surgery, or cauda equina injury. The normal periurethral EMG pattern should consist of the ability to voluntarily and reflexly contract or relax the sphincter. Anal sphincter EMG activity can normally be elicited by the ability to voluntarily tighten the anal sphincter. The EMG activity must also be interpreted in relation to activity of the detrusor muscle in response to voiding or storage of urine (CMG, flow rate). Abnormalities consist of uninhibited spincter relaxation and detrusor sphincter dyssnergia (as seen in case reports) and result in various patterns of urinary incontinence or difficulty voiding with episodes of retention respectively. These can be seen in spinal cord trauma, multiple sclerosis, cauda equina injury (acquired or congenital).

URETHRAL PRESSURE PROFILE (UPP)

UPP can be measured with various types of catheters, membrane, microtip, CO_2 catheters, and the Brown-Wickham method using running water, and each will measure different aspects of urethral function. The UPP can be measured in the supine and upright positions as the catheter is withdrawn at a constant speed from the bladder. Initially the catheter will measure the bladder pressure and then a steady increase in pressure will be noted as the catheter passes from the bladder neck to the external sphincter where a maximal pressure of 80-90 cm H_2O is reached. After this the pressure drops markedly. The distance between the bladder neck and the external sphincter represents the functional length (normally 3-4 cm) and varies with disease entities.

UPP testing is sometimes helpful in evaluation of urinary incontinence secondary to previous genitourinary surgery, stress incontinence, and sphincter dyssnergia. It is especially useful in the evaluation of the effect of an implantable incontinence device during and after surgery and in assessing sphincterotomies post-operatively.

FLOW RATE

Measurement of urinary flow is best regarded as a screening test, and can be measured electronically or simply with a stop watch and beaker. Important factors to be noted are peak flow rate, average flow rate, post-void residual, and pattern of flow rate. At least 150 to 200 cc of fluid should be voided for a good study.[4] Peak flow rate in the normal adult male should be greater than 15 cc/sec and flow below 10 cc/sec is definitely abnormal. Measurement of flow with simultaneous monitoring of EMG activity, intravesical activity, and intra-abdominal pressure can be useful in understanding the total picture since flow rate is a function of these and urethral resistance.

SUMMARY

The process of micturition is a complex one, and knowing how to best treat a specific voiding dysfunction requires proper evaluation which is now available. Previous hit-and-miss therapy can now be replaced with pharmacological and surgical manipulation tailored to a disease entity with predictable results. □

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WHY A FORENSIC AUTOPSY?*

JOEL S. SEXTON, M.D.**
GORDON R. HENIGAR, M.D.***

As consultants in forensic pathology at a medical university, the authors are often asked by police, coroners, relatives, funeral directors, doctors, lawyers, the news media, and their associates in pathology "why conduct a 'forensic' autopsy on 'this' case?" The purpose of this article is to give a better understanding to the reader of why a forensic autopsy is sometimes indicated. It should also give the reader an idea of how a forensic autopsy differs from a normal hospital autopsy and what are the benefits of doing such a procedure.

It is appropriate to mention that neither of the authors are advocating a vast increase in the number of autopsies. Even in a modern medical examiner's system, only one-third to one-fourth of the deaths are reported to the medical examiner's staff and usually only one-fourth to one-fifth of these reported deaths require autopsy. Therefore, usually only 5% to 8% of the deaths receive forensic autopsies.

What is a forensic autopsy?

A forensic autopsy is a complete postmortem exam (external physical exam of the body with appropriate photographs, charts, diagrams, tracings, and collection of physical evidence†) and necropsy (internal dissection [autopsy] with examination of each internal organ grossly and microscopically including collection of appropriate toxicology samples, and tissues for culture or special studies††). It is usually authorized by a coroner or medical examiner. A next of kin, solicitor, judge, or attorney general may also authorize such an exam.††† When authorized by the coroner, it is usually performed on a person dying a violent or sudden death in apparent good health or being found dead under suspicious or unusual conditions. The autopsy is done *in the public interest*, therefore, the family's consent is not needed since emotion or deception might govern their denial of permission on a suspected murder case.

The forensic examination differs from the normal hospital autopsy in a number of ways which center around detecting subtle homicides and documenting the findings on all cases for possible court presentation. You would expect all pathologists to do autopsies the same way, however, a survey conducted by these authors revealed that less than one tenth of the pathologists get any formal training in forensic pathology. Therefore, the detection of smothering, manual strangulation, poisoning, etc. is foreign to them. Likewise low voltage electrocution, lightning deaths, seizure deaths (natural or accident related), and similar sudden accidental deaths are difficult for them to diagnose. Conducting a scene investigation of the body which is vital to most violent deaths in forensic pathology is also not in their regimen of knowledge.

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† blood for typing; semen for typing; hair, soil, grease, glass, paint, fingernail fragments, etc for comparison, etc.

†† scanning and transmission electromicroscopy, bile and blood for typing controls, etc.

††† according to S. C. Law

Too Few Forensic Pathologists:

Therefore, because of the current method of training pathologists in this country and the deficit of forensic pathologists (354 compared to 11,530 pathologists), most pathologists would ask with sincerity, "Why should a doctor go look at a body that is strewn across the interstate highway?" "He was obviously dead and obviously hit by some vehicle" is the statement by the pathologist that usually precedes the questions — "Should I really have been able to tell the coroner if the man was dead before he was hit; drunk when he died; crossing from left to right; murdered and left in the road; had a heart attack and fell in front of the vehicle; was lying in the road; what color vehicle hit him; whether the lights were on; etc.?" These are all important questions which are usually answered by a forensic pathologist along with the collection of physical evidence from the wounds and clothing which is turned over to the police according to routine chain of evidence procedures.

Records including the autopsy protocol, charts, diagrams, tracings, evidence receipts, photographs, microscopic slides, x-rays, dental charts, etc. are kept so that the facts in the case can be clearly and accurately presented in court years later if necessary.

Cause of Death:

Cause of death is but one reason for doing a forensic autopsy. Often times since the death is a violent one, the cause of death is obvious (i.e. gunshot wound). When it is the sudden death of a healthy person or someone dying under unusual circumstances, the cause may be difficult to detect and may be dependent upon subtle findings and an accurate history (i.e., lightning, electrocution, glue sniffing, abortion, etc.).

Manner of Death:

Manner of death is an important fact determined by some forensic autopsies. The history and condition of the body at the scene are vitally important in determining manner of death yet may be very misleading particularly to a nonprofessional. Once the body has been carefully examined at the scene, moved to the morgue, re-examined, washed, re-examined, and autopsied (when necessary), the apparent manner of death may change from natural to accidental, from homicide to natural, from homicide to suicide, etc.

Some of the unwitnessed *accidental* deaths that can easily be misinterpreted as *natural* deaths if a forensic autopsy is not done include the following:

1. cold exposure
2. abortion (self induced)
3. fish bone aspiration
4. carbon monoxide poisoning
5. electrocution (low voltage without burn)
6. lightning (indoors or outdoors)
7. glue or aerosol sniffing
8. seizure secondary to a head injury
9. subdural hematoma secondary to a head injury (i.e., fall or auto accident)
10. cafe coronary (aspiration of large chunk of food)
11. delayed death following auto accident (fat emboli, pulmonary embolus, complications of rib fractures, etc.)
12. overdose due to synergistic effect of alcohol and over-the-counter and/or prescription drugs
13. etc.

It is not uncommon for unwitnessed *accidental* deaths to mimic *homicides* or *suicides.* When carbon monoxide poisoning takes a matter of minutes, the victim can stagger around and receive fairly severe injuries from falling and hitting his head. Some scenes of this sort have been described as "blood baths" and "massacres" and initially mistaken for homicides. Sexual asphyxia is often mistaken and interpreted as suicides or homicides because the victim is hanging or bound (with slip knots or locked in chains with the key attached to the lock for ready access) with a plastic bag over his head.

Some *homicides* that can easily go undetected without a forensic autopsy include the following deaths that mimic *naturals:*

1. criminal abortions
2. smothering (especially newborn)
3. manual strangulation (Figure #1)
4. soft ligature strangulation
5. a battered child
6. blunt trauma (i.e., spanking with wet towels of prisoner causing fat embolism)
7. poisoning (arsenic, insecticides, drugs, etc.)
8. etc.

Striking examples of *homicides* camouflaged as *accidents* are seen in train/pedestrian victims and auto/pedestrian or auto driver victims who reveal homicidal gunshot wounds, or more commonly blunt trauma or strangulation which the assailant hopes the vehicular trauma will obscure.

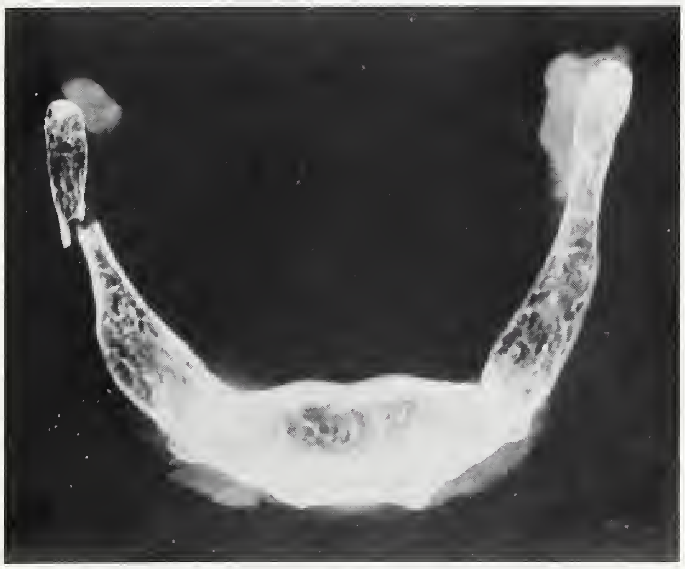

FIGURE 1: Only an autopsy can establish death by manual strangulation. Frequently one of the key findings is a fractured hyoid bone like the one illustrated above by x-ray.

A person who has five gunshot wounds to the body or two to the head is usually felt to be a *homicide*. However, finding that they were all superficial contact wounds except for one fatal wound in a depressed individual may indicate the manner to be *suicide*.

One reason to do autopsies on drivers dying in a traffic accident is to make sure a *natural* death is not misinterpreted as an *accidental* death. This may be very important if the decedent in question caused serious injury or death(s) for which his estate might be sued. It is also very important if the decedent was hit, thrown from the car, and hit again. If he was in fact dead when the second car struck him, there would be but one litigation instead of two. Some of these people die of a Berry aneurysm, brainstem hemorrhage (stroke), acute coronary insufficiency (heart attack), etc. before the accident and this causes the accident. Since their heart has already stopped beating characteristic postmortem injuries are seen instead of premortem ones.

Another area of *natural* death that is sometimes classified improperly as *homicides* is the infant usually less than one year of age that has marked anterior lividity (resembling contusions), bloody postmortem ooze from the mouth and nose (resembling injury) and diaper rash. These infants at autopsy have no injury and die from a mysterious killer called the Sudden Infant Death Syndrome which the medical profession does not yet understand. However, medicine does consider these as natural deaths since no injuries are found. Unfortunately, adult resuscitation technique can produce postmortem internal hemorrhage due to lacerated bowel, liver, etc. and mimic premortem injury in these young infants causing confusion at autopsy.

Circumstances Surrounding Death:

The circumstances surrounding a death constitute a very important reason for doing a forensic autopsy. These represent the facts found at autopsy which refute or confirm the statements of witnesses.

These facts and their interpretation often times change a self defense case into cold-blooded murder.

Examples include the husband who said his wife who was sitting across the room was accidentally shot when his pistol discharged while he was cleaning it. The autopsy and ballistics findings regarding the powder burns on the decedent's forehead (Figure #2) showed she was 9 inches from the muzzle when the gun discharged. He would have to have been sitting in her lap while cleaning the gun.

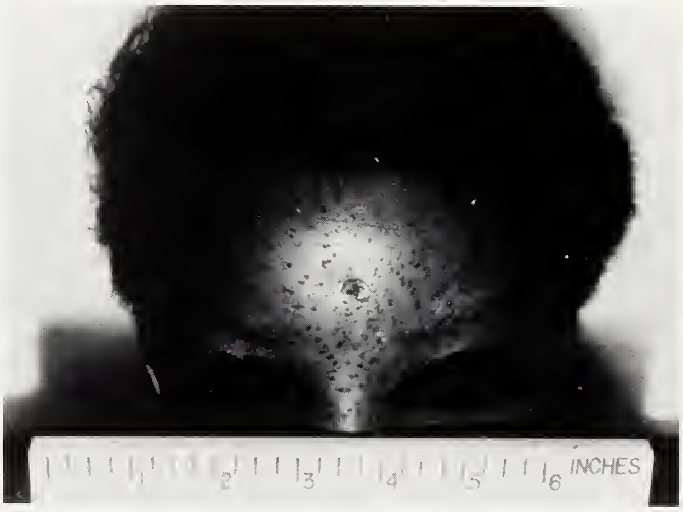

FIGURE 2: This gunshot wound of the forehead with the four inch powder burn pattern indicated the murder weapon was 9 inches from the victim. The husband who said he was cleaning his gun across the room was convicted of murder.

A young, 11 year old boy had a gunshot wound in the back of his neck that looked like it exited in the front. According to his father, he was also shot accidentally while the father was cleaning the pistol. Examination by the forensic pathologist revealed the bullet entered the front of the neck; it was a contact wound with soot, charring of the skin, splitting of the skin, carbon monoxide change in the muscle, and powder particles; and the wound in the back of the neck represented a "shored" exit wound due to the loose collar being pulled tightly against the neck by a hand gripping the front of the shirt neck. The boy's body also showed prior trauma at autopsy.

These are circumstances where the sequence of gunshot wounds can show that a case could not be self defense.

A husband was shot by his frail wife six times with an automatic pistol. She claimed she did so because he was about to hit her with a judo chop. The defense was impressed with the fact that one of the shots went thru the decedent's hand. However, when it was shown that the first four shots entered the decedent's left neck and shoulder from above while he slept in a TV chair and one of the last two went through his hand while he lay partially paralyzed on the floor, it was clear that it was murder. One of the last two bullets which followed a parallel path in the body struck one of the first four bullets which also followed a different parallel path. The only bullet that exited (based on the six empty cases that were found) passed through a sweater on the back of the TV chair and entered the wall after passing through the neck indicating the sleeping position of the decedent when first shot.

A similarly unique case occurred when a man shot in the abdomen and the back with a shotgun was autopsied. It turned out that a single shot from the shotgun blast to the abdomen penetrated the base of the plastic wadding (Figure #3) from the shotgun wound of the back showing the man was first shot in the back.

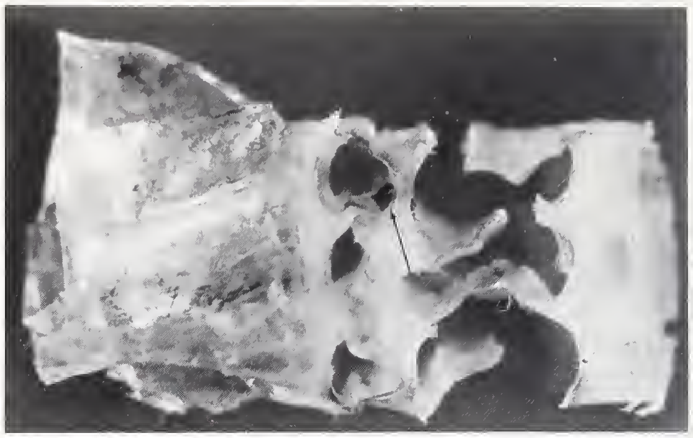

FIGURE 3: This plastic shotgun wadding which was recovered at autopsy had a pellet embedded in its base. The fact that a pellet from the second shotgun blast was found embedded in the wadding from the first blast showed the sequence of the blasts. The fact that the decedent was first shot in the back convicted the suspect of murder in spite of his self defense plea.

Forensic autopsies also exonerate the innocent. The following case illustrates this point.

A black man was brought to the ER with 28 gunshot wounds. The rumors of police brutality rapidly spread. However, when the forensic autopsy findings were made public, it was clear that the decedent who had just killed his wife and mortally wounded his mother-in-law was fired at one time each by three law officers who were sent to apprehend this known felon. Two of the law officers used shotguns with no. 4 buckshot (22 shot in a shell) and one used a machine gun that hit the decedent twice during one blast. The autopsy also revealed that the decedent was lying on his back on the ground when the last shot was fired at him. However, he was aiming his 44 magnum revolver at the law officer who shot him, according to the shot pattern on his right hand, arm, and the pistol.

Clues to a Suspect:

An important reason for doing a forensic autopsy is the detection of clues to the suspect. Most of these clues simply eliminate certain suspects (i.e., if black hair is found on the body of a rape-murder victim, the suspect is probably not a blonde, or brunette *unless* a wig was worn which shed). The type deaths that lend themselves to clues are rape murders, hit and runs, beatings, stabbings, and gunshot wounds where blood, semen, hair, fibers, saliva, patterns of injury on skin (i.e., impression of wrench in beating), etc. can be collected as evidence. Items like broken fragments of fingernails and bite marks (Figure #4) represent unique evidence which can be traced to only one suspect like fingerprints.

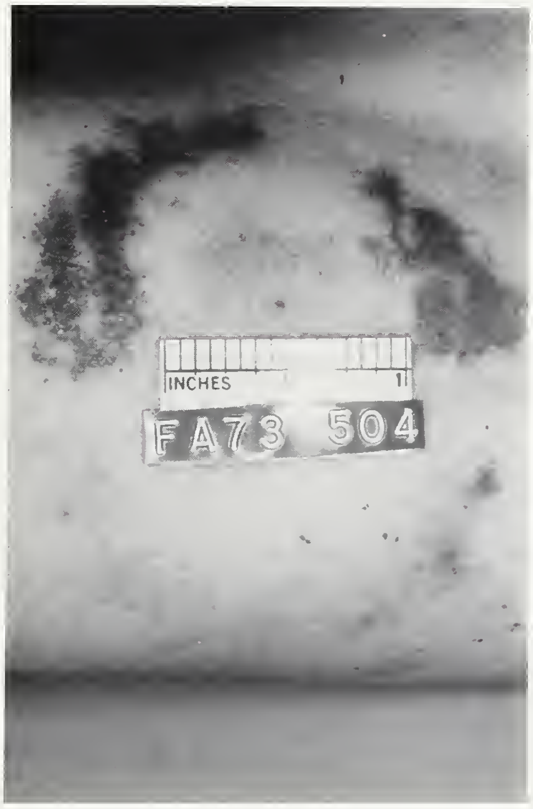

FIGURE 4: Bite mark showing missing upper right central and right lateral incisors. The missing teeth and other features made the bite unique to one suspect.

Body Found in Unusual Location or Condition:

Other reasons for a forensic autopsy include finding the body in an unusual location or condition. Some of these situations include:

1. the pilot of a plane
2. driver of an unexplained auto accident
3. in police custody (detect mistreatment or refute it)
4. at work (workmen's compensation)
5. in public building without history of serious disease or illness (to detect hazard and prevent needless suit)
6. burned, charred, skeletonized, or decomposed body (to detect homicide and to identify decedent properly)
7. beside road, railroad track, etc. (to detect homicide vs. natural or accidental death)
8. born dead unattended by physician (to detect infanticide)
9. died sudden in apparent good health especially young infant or child (to detect homicide, contagious disease, and congenital defects)
10. dying suddenly within one year of serious injury — auto accident, fall, etc. (to determine if death is related to injury, i.e., seizure, pulmonary embolus, etc.)
11. being found dead in garage, garden, near ladder, near electrical appliance, etc.

Detection of Hazards to the Public:

In some jurisdictions, the following area is a bonus, in others, it is a principal reason for doing a forensic autopsy. This is the area of recognition of public hazard and other conditions requiring private or public education.

Some of the public hazards requiring public education as a remedy include:

1. electrocutions
2. power lawn mower deaths (projecting rocks, etc. like bullets)
3. driver/passenger death (due to not using seat belts)
4. lightning deaths (may respond to delayed resuscitation)
5. aerosol or glue sniffing deaths (fibrillation caused by Freon and toluene are now prevented by "pump" sprays and non-toxic glues)
6. deaths due to improperly repaired cribs or baby beds
7. boating deaths (where life preservers and kill switches were not used)
8. traffic deaths related to alcohol
9. overdose deaths due to alcohol plus over-the-counter and/or prescription drugs
10. battered child deaths (parents rarely psychotic and can be helped)
11. drownings in sandpits, etc.

Some deaths magnify the need for specific action. These include deaths related to bridge fishing (construction of catwalk for fishermen); suffocation due to baby rattle (action by Consumer Product Safety Division to ban certain product designs); deaths due to inability to open child-proof caps (similar action as above by Consumer

Products Safety Division); deaths in motorcyclists who are only half drunk legally (new legislation on legal drunkeness level in cyclists who need faster reflexes than auto drivers); deaths on recreational rides (enforcement of present laws or legislate better ones); deaths due to improper working conditions (establish regulations to improve working conditions); carbon monoxide deaths (where the source must be found and eliminated); etc.

Some deaths indicate education or counseling of parents or spouse of decedent. Included in these are "crib deaths" (Sudden Infant Death Syndrome) — where the wife and husband blame themselves for a death that even medical science cannot yet prevent; congenital defect deaths — where the family should be counseled as to having more children; contagious disease deaths — where all contacts should be checked and followed by a physician; and sickle cell trait as an incidental or death related finding — where the family should be alert to the need for having blood relatives checked and counseled if they possess the trait.

Conclusions:

In general, the forensic autopsy is a tool for confirming the obvious causes of death, documenting facts for presentation in court, determining the subtle causes of death (especially in homicidal cases), helping to determine the manner of death, finding facts about the circumstances surrounding death, and clues to the suspect. As a bonus in some jurisdictions, hazards to the public are recognized.

Basically, it requires experience and a suspicious mind working *in the public interest* to know when a forensic autopsy is indicated. The reasons for doing a forensic autopsy include the obtaining of many facts such as the presence of a contagious disease, public hazard, or other facts which might aid in the proper ajudication of a given murder charge; prevention of an epidemic; recognition and correction of a public health hazard; prevention of a needless, costly law suit; prevention of false accusations or charges; recognition of areas requiring public education; and in general, the orderly handling of the consequences of violent or sudden deaths through the use of *facts not fantasy.* □

Tenuate® Ⓒ IV
(diethylpropion hydrochloride NF)

Tenuate Dospan®
(diethylpropion hydrochloride NF) controlled-release

AVAILABLE ONLY ON PRESCRIPTION
Brief Summary
INDICATION: Tenuate and Tenuate Dospan are indicated in the management of exogenous obesity as a short-term adjunct (a few weeks) in a regimen of weight reduction based on caloric restriction. The limited usefulness of agents of this class should be measured against possible risk factors inherent in their use such as those described below.
CONTRAINDICATIONS: Advanced arteriosclerosis, hyperthyroidism, known hypersensitivity, or idiosyncrasy to the sympathomimetic amines, glaucoma. Agitated states. Patients with a history of drug abuse. During or within 14 days following the administration of monoamine oxidase inhibitors, (hypertensive crises may result).
WARNINGS: If tolerance develops, the recommended dose should not be exceeded in an attempt to increase the effect; rather, the drug should be discontinued. Tenuate may impair the ability of the patient to engage in potentially hazardous activities such as operating machinery or driving a motor vehicle; the patient should therefore be cautioned accordingly. *Drug Dependence:* Tenuate has some chemical and pharmacologic similarities to the amphetamines and other related stimulant drugs that have been extensively abused. There have been reports of subjects becoming psychologically dependent on diethylpropion. The possibility of abuse should be kept in mind when evaluating the desirability of including a drug as part of a weight reduction program. Abuse of amphetamines and related drugs may be associated with varying degrees of psychologic dependence and social dysfunction which, in the case of certain drugs, may be severe. There are reports of patients who have increased the dosage to many times that recommended. Abrupt cessation following prolonged high dosage administration results in extreme fatigue and mental depression; changes are also noted on the sleep EEG. Manifestations of chronic intoxication with anorectic drugs include severe dermatoses, marked insomnia, irritability, hyperactivity, and personality changes. The most severe manifestation of chronic intoxications is psychosis, often clinically indistinguishable from schizophrenia. *Use in Pregnancy:* Although rat and human reproductive studies have not indicated adverse effects, the use of Tenuate by women who are pregnant or may become pregnant requires that the potential benefits be weighed against the potential risks. *Use in Children:* Tenuate is not recommended for use in children under 12 years of age.
PRECAUTIONS: Caution is to be exercised in prescribing Tenuate for patients with hypertension or with symptomatic cardiovascular disease, including arrhythmias. Tenuate should not be administered to patients with severe hypertension. Insulin requirements in diabetes mellitus may be altered in association with the use of Tenuate and the concomitant dietary regimen. Tenuate may decrease the hypotensive effect of guanethidine. The least amount feasible should be prescribed or dispensed at one time in order to minimize the possibility of overdosage. Reports suggest that Tenuate may increase convulsions in some epileptics. Therefore, epileptics receiving Tenuate should be carefully monitored. Titration of dose or discontinuance of Tenuate may be necessary.
ADVERSE REACTIONS: *Cardiovascular:* Palpitation, tachycardia, elevation of blood pressure, precordial pain, arrhythmia. One published report described T-wave changes in the ECG of a healthy young male after ingestion of diethylpropion hydrochloride. *Central Nervous System:* Overstimulation, nervousness, restlessness, dizziness, jitteriness, insomnia, anxiety, euphoria, depression, dysphoria, tremor, dyskinesia, mydriasis, drowsiness, malaise, headache; rarely psychotic episodes at recommended doses. In a few epileptics an increase in convulsive episodes has been reported. *Gastrointestinal:* Dryness of the mouth, unpleasant taste, nausea, vomiting, abdominal discomfort, diarrhea, constipation, other gastrointestinal disturbances. *Allergic:* Urticaria, rash, ecchymosis, erythema. *Endocrine:* Impotence, changes in libido, gynecomastia, menstrual upset. *Hematopoietic System:* Bone marrow depression, agranulocytosis, leukopenia. *Miscellaneous:* A variety of miscellaneous adverse reactions has been reported by physicians. These include complaints such as dyspnea, hair loss, muscle pain, dysuria, increased sweating, and polyuria.
DOSAGE AND ADMINISTRATION: Tenuate (diethylpropion hydrochloride): One 25 mg. tablet three times daily, one hour before meals, and in midevening if desired to overcome night hunger. Tenuate Dospan (diethylpropion hydrochloride) controlled-release: One 75 mg. tablet daily, swallowed whole, in midmorning. Tenuate is not recommended for use in children under 12 years of age.
OVERDOSAGE: Manifestations of acute overdosage include restlessness, tremor, hyperreflexia, rapid respiration, confusion, assaultiveness, hallucinations, panic states. Fatigue and depression usually follow the central stimulation. Cardiovascular effects include arrhythmias, hypertension or hypotension and circulatory collapse. Gastrointestinal symptoms include nausea, vomiting, diarrhea, and abdominal cramps. Overdose of pharmacologically similar compounds has resulted in fatal poisoning, usually terminating in convulsions and coma. Management of acute Tenuate intoxication is largely symptomatic and includes lavage and sedation with a barbiturate. Experience with hemodialysis or peritoneal dialysis is inadequate to permit recommendation in this regard. Intravenous phentolamine (Regitine®) has been suggested on pharmacologic grounds for possible acute, severe hypertension, if this complicates Tenuate overdosage.

Product Information as of April, 1976

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References: 1. Citations available on request — Medical Research Department, MERRELL RESEARCH CENTER, MERRELL-NATIONAL LABORATORIES, Cincinnati, Ohio 45215. 2. Hoekenga, M.T., O'Dillon, R.H., and Leyland, H.M.: A Comprehensive Review of Diethylpropion Hydrochloride. International Symposium on Central Mechanisms of Anorectic Drugs, Florence, Italy, Jan. 20-21, 1977.

Merrell

WHY ARE SOCIAL SECURITY COSTS SOARING?

FRED H. FELLERS, M.D.*

There are many aspects to the Social Security Program, and the answer to the question of soaring costs would depend upon the aspects of the program being reviewed. Since the Social Security Program was established in 1936, the number of workers covered has steadily increased and many of the workers who were just beginning their working careers in 1936 are now reaching retirement age. Today there are approximately 130 million people covered for benefits as workers, survivors, or dependents and they look to the Social Security Administration for a range of services that could not have been foreseen in the days of the "New Deal."

In 1954, Congress addressed the issue of providing some measure of support to individuals whose working lives had been cut short due to disability. Since that time expanded coverage and amendments to the Act have greatly increased the number of recipients. The following table lists the type of disability beneficiaries and the total monthly payments received in South Carolina as of December, 1977:

Category	*Number*	*Monthly Payments*
Disabled Workers	47,868	$11,583,000.00
Spouses of Above	7,988	552,000.00
Children of Above	25,321	1,738,000.00
Total	81,177	$13,873,000.00

In the Disability Program, the physicians in our state are directly involved, and it is this program that will be focused on here.

The Social Security Disability Program is the largest disability program in the United States. Effective administration of the program depends on the active cooperation and participation of the medical profession. Physicians participate in many ways in assuring the medical soundness of the program and in helping their patients receive the benefits to which they may be entitled. In addition, the integrity of the program is protected through accurate reports that ensure that only those who are truly disabled receive benefits.

Under the Social Security Act disability is an administrative term defined as: "Inability to engage in *any* substantial activity by reason of any medically determinable physical or mental impairment which is expected to result in death, or has lasted or can be expected to last for a continuous period of not less than 12 months."

A medically determinable impairment is further defined as a medically *demonstrable* anatomical, physiological, or psychological abnormality manifested by signs or laboratory findings apart from symptoms. Abnormalities which manifest themselves only as symptoms are not medically determinable. It is this definition which leads us into a discussion of one of the most costly aspects of the Disability Program.

The file of every applicant for Disability Insurance Benefits must contain medical reports signed by a duly licensed physician. Such a report should contain the applicant's medical history relating to the impairment or impairments which prevent work and should contain a description of a physical examination and such supporting laboratory data needed to determine the nature and severity of the impairment. All symptoms, signs, and laboratory findings which have a bearing on the impairment should be reported. Disability decisions cannot be made on the basis of clinical opinions relating to an applicant's diagnosis, prognosis, or remaining capacity to work unless the supporting signs or laboratory findings are also reported. In other words, the completeness of the report should be such that any physician reviewing it could arrive at a definite conclusion regarding the type and severity of the impairment or impairments, and the restrictions they place on the individual's daily activities. Certainly the treating physician may not have such detailed information on each of his patients, and the program is set up to identify sources that can be of assistance to the claimant in providing information.

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When an applicant applies for disability benefits, a list of all medical treatment sources is obtained and a proper "release of information" form is signed by the claimant. The Disability Determination Division contacts the sources, (private physician, clinics or hospitals) for medical information. This contact may be by telephone or written request depending upon the source's preference. It has been estimated that 75% of all claims could be decided on evidence of record if the Disability Division could obtain this information in a complete and timely manner. It is realized that the physician receives many requests daily for medical records, and in order to assist with requests from the Disability Determination Division, a tele-dictation system is available 24 hours a day, seven days a week, on which these reports may be dictated. Typists transcribe the dictation from the machine, make two copies, and return them to the physician for review, correction, and signature. The original is returned to the Disability Division and a copy may be retained by the physician for his files. Even when the treating physician's medical information is limited, a report showing what additional information is needed may help direct subsequent development.

The report may allow the Disability Division to secure a consultative examination that is more directly related to the actual information needed to properly evaluate a particular impairment and — as a by-product — provide the treating physician with valuable information on his patient.

The handbook for physicians entitled "Disability Evaluation Under Social Security" is most helpful in outlining the type of information needed under the program regulations. These have been distributed to physicians throughout the state, and may be obtained by calling or writing the Disability Determination Division.

At the present time there is a shortage of practically all specialists willing to perform consultative examinations for the Disability Division. As an example, a recent survey revealed only one or two internists are performing examinations in several of South Carolina's larger cities. This results in a delay of four to five weeks in processing claims for needy individuals. It also causes a marked increase in the administrative cost of the program. There is a need for assistance from not only Internists, but all specialists, including Certified Family Practitioners, and General Practitioners.

CONCLUSIONS:

We are all aware of the fact that the cost of the Social Security Program is soaring and there are many aspects over which we have no control. However, in the disability aspect of the program, the physicians can make an important contribution toward keeping the cost down.

There are several important ways that physicians can help contain the cost of the disability programs:

(1) Be familiar with the requirements of the Disability Programs to be able to advise your patients as to when and how to apply for disability benefits. The physician's handbook entitled "Disability Evaluation Under Social Security" is available and outlines the information required for making a decision of disability.

(2) Reply promptly to request for medical information on your patients. Undue delay requires follow-up letters and telephone contacts that are costly and, therefore, adds to the cost of the program, as well as adding to the number of interruptions your office experiences.

(3) Furnish as complete records as possible following the guidelines in the Physician's Handbook. Naturally, at times the attending physician's records may not contain information such as EKG's, x-rays, or laboratory studies. By noting in your report that these studies have not been done but are needed, the Disability Division upon review of the report, may authorize and pay for the needed work.

(4) Contribute to the professional effectiveness of the program by examining applicants for the

Disability Determination Division. Often claimants do not have a source of medical treatment, and it is necessary to secure an examination. At the present time the number of physicians throughout the state participating in disability evaluations is quite small in comparison to the number of physicians practicing in South Carolina. This naturally leads to delays in scheduling examinations, receiving the reports, and puts a heavy burden on those physicians participating. With more physician participation in the program, the impact would be more evenly distributed and would result in prompt examinations and reports, eliminating delays, and reducing costs.

(5) The disability programs under the Social Security Administration cannot be effectively administered without the complete cooperation and assistance of physicians. Any cost saving procedures, as noted above, directly affects physicians as taxpayers, since these programs are supported by the Social Security Trust Fund and general tax revenue. While we are vitally interested in the cost factor, we must not lose sight of the main goal of the programs. That is, to make fair and just decisions in a timely manner to assure that those individuals entitled to disability benefits receive them, and those not entitled are denied benefits. This is only possible with your help and cooperation.

If you are interested in performing examinations, have any questions, or desire additional information, please contact the Disability Determination Division, State Office, P. O. Box 4945, Columbia, South Carolina 29240. □

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President's Page

THE ACTION BEGINS

Charleston: January 8, 1979

This is the week — and the month — of new beginnings in Government. I am particularly mindful of this as I plan to go tomorrow to Columbia, first to make the SCMA presentation to the Congressional Open Forum, and then to act as Doctor of the Day as the Legislative Session opens.

My presentation to our Congressional Delegation will be brief, touching on only three of the major problems which affect the medical profession nationally. These are the Federal Trade Commission's activities, the Cost Containment issue, and National Health Insurance. The SCMA leadership hopes to have the opportunity to meet at a later date with our Congressmen in Washington for a less formal and more in-depth discussion of these and other issues.

The Doctor of the Day Program is now firmly established, not only as a worthwhile service to Legislators at the State House, but also as a channel whereby physicians from all over the state can become better acquainted with their own representatives, can demonstrate their interest in critical state issues, and can view first hand the process of State Government in action. I feel sure that the program will be over-subscribed again this year and that there will be more applicants for Doctor of the Day than there are days available. If you are genuinely interested, I hope you have sent your application early.

Later this week, the inaugural festivities will take place and Governor Richard W. Riley and Lieutenant-Governor Nancy Stevenson will assume their very important responsibilities and will begin their administration of the affairs of state. I congratulate them and wish them well. They have many personal friends in the medical profession, and they both have shown a keen interest in health care issues in the Legislature. I feel they will be attentive to health and medical problems during the next four years.

The interest and active participation of physicians all over the state in the political process during the recent elections was most gratifying. It must be remembered now, however, that the ballgame is just beginning. So far, we have just chosen the players. During the coming months many issues will relate to medicine directly or indirectly and will require input, and hopefully direction, from our profession. Your Council, Officers and Legislative Committee, as well as the SCMA staff, will be working constantly to keep abreast of the issues (or even ahead of some of them!). But we will need continuing help from members over the entire state. I urge you to become well acquainted with your Legislators, to communicate regularly if you have an "in" with one or more of them, and then to be prepared to follow through promptly and effectively when you are called upon to address a specific issue.

Remember, the future of medicine is in *your* hands, too!

John C. Hawk, Jr., M.D.
President

SOUTH CAROLINA MEDICAL ASSOCIATION

AUXILIARY

WHO IS RESPONSIBLE FOR REPORTING CHILD ABUSE UNDER THE NEW LAW?

THE CHILD PROTECTION ACT OF 1977 (R297, H2796)

To Provide Penalties For Violations And To Repeal Act 1068 Of 1972 Relating To The Protection Of Children Under The Age Of Eighteen From Certain Physical Abuse.

PURPOSE: Recognizing that abused and neglected children (a person under eighteen whose physical or mental health or welfare is harmed or threatened with harm) in South Carolina need protection, it is the purpose of this act to save them from injury and harm by establishing an effective reporting system and encouraging the reporting of children in need of protection: . . .

PERSONS TO REPORT: Section 5 (A) Any physician, nurse, dentist, optometrist, medical examiner or coroner, or any other medical, mental health or allied health professional, Christian Science practitioner, religious healer, school teacher or counselor, social or public assistance worker, child care worker in any day care center or child caring institution, police or law enforcement officer, or any judge having reason to believe that a child's physical or mental health or welfare has been or may be adversely affected by abuse or neglect is required to report or cause a report to be made in accordance with this section.

PHOTOGRAPHS AND X-RAYS: Section 7. Any person required to report under Section 5 of this act may take, or cause to be taken, photographs of the areas of trauma visible on a child who is the subject of a report and, if medically indicated, cause to be performed a radiological examination of the child without the consent of the child's parents or guardians. . . . All photographs and x-rays taken, or copies of them, shall be sent to the appropriate local child protective service agency or county department of social services at the time a report pursuant to Section 5 is made, or as soon thereafter as possible.

ABROGATION OF PRIVILEGED COMMUNICATION: Section 10. The privileged quality of communication between husband and wife and any professional person and his patient or client, except that between attorney and client or priest and penitent, is abrogated and shall not constitute grounds for failure to report or the exclusion of evidence in any civil child protective proceeding resulting from a report pursuant to this act.

PENALTIES: Section 19. Any person required to report a case of child abuse or neglect, or any person required to perform any other function under this act, who knowingly fails to do so shall be deemed guilty of a misdemeanor and upon conviction shall be fined not more than five hundred dollars or be imprisoned for not more than six months, or both.

APPROVED THE 13th DAY OF JUNE 1977 BY JAMES B. EDWARDS, GOVERNOR.

by Sara Shingler
Auxiliary Chairman
Child Protection

Editorials

BEEPERS

"Daddy, are you *really* going to throw your beeper in the ocean?" Thus, recently, spoke my young child with a gleam in her eyes reflecting the sagacity of youth. She must have realized that the tiny object with the curious emissions held a great power over its possessor. Although weighing only 550 grams and measuring only 11.2 by 3.3 by 1.9 centimeters, it held beneath its bland plastic-and-aluminum exterior the ability to make one freeze in his tracks — instantly.

Beepers find people — thus run the advertisements. The purpose of this editorial is to suggest that, also, *beepers make problems*. It is time for some solutions.

Now, admittedly, the kind of person who would volunteer to edit a medical journal would place special value on the privacy of his thoughts and would therefore tend to assume an adversary relationship with any object armed with such powers. There are those in our profession who *thrive* on constant interruption. And, after all, beepers are but an extension of a previous invention — the telephone — and thus the concept of "instant access" is hardly new.

But the telephone itself was a mixed blessing. The late Dr. Waring, who edited this journal for 17 years, reflected on the medical scene in South Carolina in 1876 as follows:

> "This was a significant year, for the introduction of the telephone was to produce a great change in medical practice, adding tremendously to the physician's range and usefulness, *but destroying forever whatever opportunity for fruitful contemplation had been inherent in the former slowness of communication.* (italics mine.)[1]

It is possible, however, to create buffers between oneself and the telephone. Not so with beepers — at least as commonly used.

The rapidity with which beepers have become a part of our daily lives is astonishing. My dictionary, which I prize for its completeness of current idiomatic speech, including slang, doesn't even contain the word "beeper." I do not recall seeing one ten years ago — now they are everywhere, and some physicians carry several of them. Even more than the stethoscope, the beeper seems to identify its bearer as "a doctor" although, as with the stethoscope, there are others who go about similarly equipped. They go off everywhere: in rooms, in corridors, in automobiles, and in the most private of places. Their screeches and groans are a part of our lives, yet always bring a startle. Like the thrill evoked by the whir of a rising covey of quail, they rivet one's attention every time, however accustomed one has become to the phenomenon.

As usual, Dr. Franz Ingelfinger put it best. Writing recently about "the graying of grand rounds," comparing the decorum of the past with the chaos of the present, he noted:

> ". . . the most erosive feature of today's Grand Rounds is the devastating din, with its most relentless component, the *insistent beeper*. At intervals of 90 ± 15 (I S.D.) seconds, someone's beeper *growls and squeals*, to be followed by a croaking voice demanding the attention of this or that member of the audience. . . . 'In the recognition of thyroid dysfunction,' the speaker says, 'the most crucial. . . .' — '*Beep, blab, wheep*,' erupts some beeper, and whatever is 'most crucial' in the speaker's mind remains a mystery forever to those who entered the hall to hear ideas, not cacophony. . . . (italics mine).[2]

Dr. Ingelfinger's reflections about Boston certainly hold true in Columbia, and the most hopeful note is that, in one Southern city, it is rumored that a powerful chief of medicine has declared that at *his* rounds beepers must be "checked in," like overcoats at a good restaurant, before one can enter to enjoy the feast.

Like the telephone, the beeper is an imperfect invention — not so much in its concept or even in its design, but rather in its operation. Paramedical personnel of every description naturally tend, when in doubt, to "beep the doctor" rather than to study his progress notes or to *look up* necessary information in the hospital formulary, *Physician's Desk Reference*, or other readily-available sources. The switchboard operators of hospitals and answering services, like the rest of us, become too busy at times and ring the wrong person or — even worse — may forget that one has called in and advised that the beeper has been turned "off" in favor of the telephone, thereby resulting in a missed message. Like any good invention, overuse causes problems.

The time may have come for an *opinion poll* about beepers. The *emergency beep*, we would all agree, is useful, desirable, and necessary. I suggest that *over-beeping* is seriously eroding our ability to exercise our one necessary purpose: thoughtful *decision-making* in the interests of our patients.

Thoughtful decision making requires time, and the fewer interruptions, the better. The rendering of compassion also requires time, and again, it is questionable whether over-beeping can fail to affect adversely our ability to carry out these functions. Over-stimulated, we become creatures of conditioned reflexes. Our ability to render *individualized* concern suffers.

Imprinted upon our collective nostalgia for a bygone era of slower pace is Sir Luke Fildes' famous painting, "The Doctor." A concerned and somber physician keeps vigilant watch over a sick child. In the background, the child's mother weeps while the father tries to comfort her. The painting symbolizes the doctor's care and compassion. I kept a fine reproduction of this painting in my study for a number of years and studied it frequently. But like any great painting, there is always something else to see in it. Recently, I learned that I had never given proper attention to one important detail — a small, crumpled piece of paper in the foreground.

The story goes that the physician had gone out to visit the farm family in the midst of an influenza epidemic. The child seemed to be near death, and he chose to stay and keep his vigil. Someone knocked at the door and presented a message to be given to the physician. He opened the message, read it, and without moving an eyebrow clenched it in his hand and tossed it on the floor. The child survived, and the doctor left the next morning. Legend has it that the farm family picked up the message from the floor, and learned that it had told the doctor that his own child had died that night.

Could this event have occurred today? I think not. It is not so much that emergency room visits have replaced house calls, but rather that instant access to verbal communication would have *commanded* the physician to divert his attention from the child in front of him.

There are a number of possible solutions. We could revert to placing primary reliance on telephone answering services, with which we would check in periodically. Beepers would be used for emergency calls only. We could insist that non-emergency beeps be placed only at every hour or half-hour, and we could allot suitable periods to respond to their messages. We could do more to request individualized beeping patterns, to suit our individual practices and needs. As the number of physicians increases, the impulse to be "always instantly accessible" may grow stronger, and thus beepers with their many imperfections may become an even greater part of our lives. The time to consider their implications for our effectiveness and for our lives may be *now*.

Whoops . . . there goes that darn thing again!

— CSB

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THE PULSATILE ABDOMINAL MASS: AN APPROACH TO DIAGNOSIS*

STEPHEN I. SCHABEL, M.D.**
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GERALD M. RITTENBERG, M.D.**

The discovery of a pulsatile abdominal mass on physical examination is alarming to both the patient and the physician. There are many causes, but because of its high mortality and morbidity, aneurysm of the abdominal aorta should be diagnosed or excluded as rapidly and with as little patient risk, discomfort and expense as possible. The purpose of this paper is to present an analysis of a series of patients seen over the last two years at the Medical University of South Carolina with pulsatile abdominal masses, to stress the importance of ultrasound in their work-up and to present a logical approach to their imaging evaluation.

PATIENT MATERIAL

From January, 1976 through January, 1978, 120 patients were referred to the ultrasound section of the Department of Radiology at the Medical University of South Carolina for evaluation of pulsatile abdominal masses to rule out abdominal aortic aneurysm (AAA). AAA was defined as a focal dilatation of the aorta of 3cm or more.

Of the total group of 120 patients, 60 (50%) were shown to have dilatation of the aorta in excess of 3cm; the remainder had normal caliber aortas and were either thin with a prominent lumbar lordosis or had adjacent organs between the aorta and the abdominal wall (Fig. 1A, B). The age range was from 36 to 87 years (average 63); those with aneurysms were slightly older (range 52 to 87, average 65) than those without (range 36 to 82, average 61). Seventy (58%)were male and 50 (42%) female. The male predominance was even greater in those with aneurysms (63% male, 37% female). Nearly equal numbers were black (57) and white (63).

Most (82%) of those shown to have normal aortas were asymptomatic; six (10%) had abdominal pain, three (5%) had a bruit and two (3%) had peripheral vascular disease. More of those with abdominal aortic aneurysms had symptoms, but most (40, 67%), were also asymptomatic. Pain was present in 14 (23%); three each (5%) had a bruit or peripheral vascular disease.

Plain abdominal films showed the aneurysm in only 29 (48%). Ultrasound was very accurate in defining the AAA, measurement by ultrasound being + or − 0.5 cm of the measurement at surgery in all those operated upon. Mural thrombus was shown in only 23 (38%) aneurysms but was present in 18 (64%) of the 28 over 5 cm in diameter. Mural thrombus visible by ultrasound was found at surgery in all those operated upon.

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Angiography was performed in 19 and demonstrated the abdominal aortic aneurysm in all except two, both with extensive mural thrombus. Renal artery involvement was present in one. Surgery was performed in 21 (35%) patients all but four of whom had AAA greater than 5cm in diameter. These four had symptoms associated with 4-5cm AAA. The remainder of the patients, many with large aneurysms, were not operated upon because of underlying cardiac, CNS or neoplastic disease.

DISCUSSION

Abdominal aortic aneurysm is an infrequent disease, but as more individuals live to older ages, degenerative atherosclerotic disease and aneurysms are likely to be encountered more frequently. While some patients will present with abdominal or back pain, most will be asymptomatic when discovered to have a pulsatile abdominal mass. Rapid and accurate diagnosis is important, because if left untreated AAA carries a significant morbidity and mortality. The risk of rupture increases rapidly in aneurysms over 5cm in diameter and only 50% of patients with AAA over 6cm in diameter survive one year.[4] Still larger aneurysms have an even worse prognosis. Modern surgical techniques have lowered the elective operative mortality well below 5%[5] in experienced hands but the mortality once rupture has occurred remains over 60%.[2]

At least half of all pulsatile abdominal masses will not prove to be AAA, and particularly in thin lordotic individuals the normal aorta may be palpated directly or by transmitted pulsations through adjacent organs (Fig. 1A,B). As a result diagnostic imaging procedures should, if possible, avoid painful and expensive tests and those that carry a significant risk to the patient. A suggested outline of the imaging evaluation of a pulsatile abdominal mass is shown in Figure 3.

Plain radiographs of the abdomen, because they are readily available, inexpensive and free of significant risk or discomfort to the patient, are very useful. When the aorta is extensively calcified and normal in caliber, aneurysm can be excluded. If the aneurysm is present and extensively calcified, it can be measured accurately allowing for radiographic magnification. Occasionally a non-calcified aneurysm will be visible as a soft tissue mass. Only about 60% of abdominal aortic aneurysms will be visible on plain films[3] and when the aorta is not visible, further

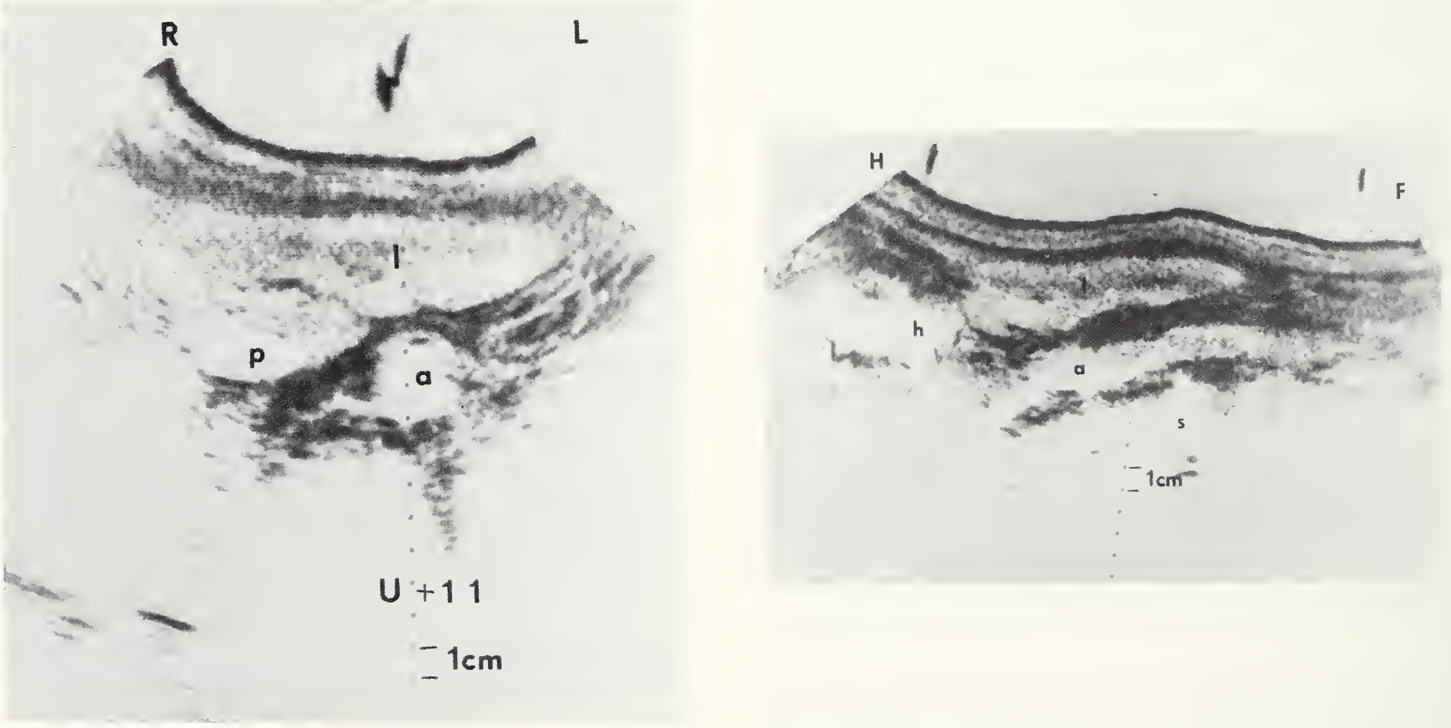

FIGURE 1. 74 year old female with pulsatile abdominal mass.

a. Transverse scan 11 cm above the umbilicus showing a normal aorta (A) 2 cm in diameter. Prominent left lobe of liver (L) and a normal portal vein (P)

b. Longitudinal scan on the same patient again showing a normal aorta (A), prominent liver (L). Blood filled heart (H) and shadow from the spine (S) are visible.

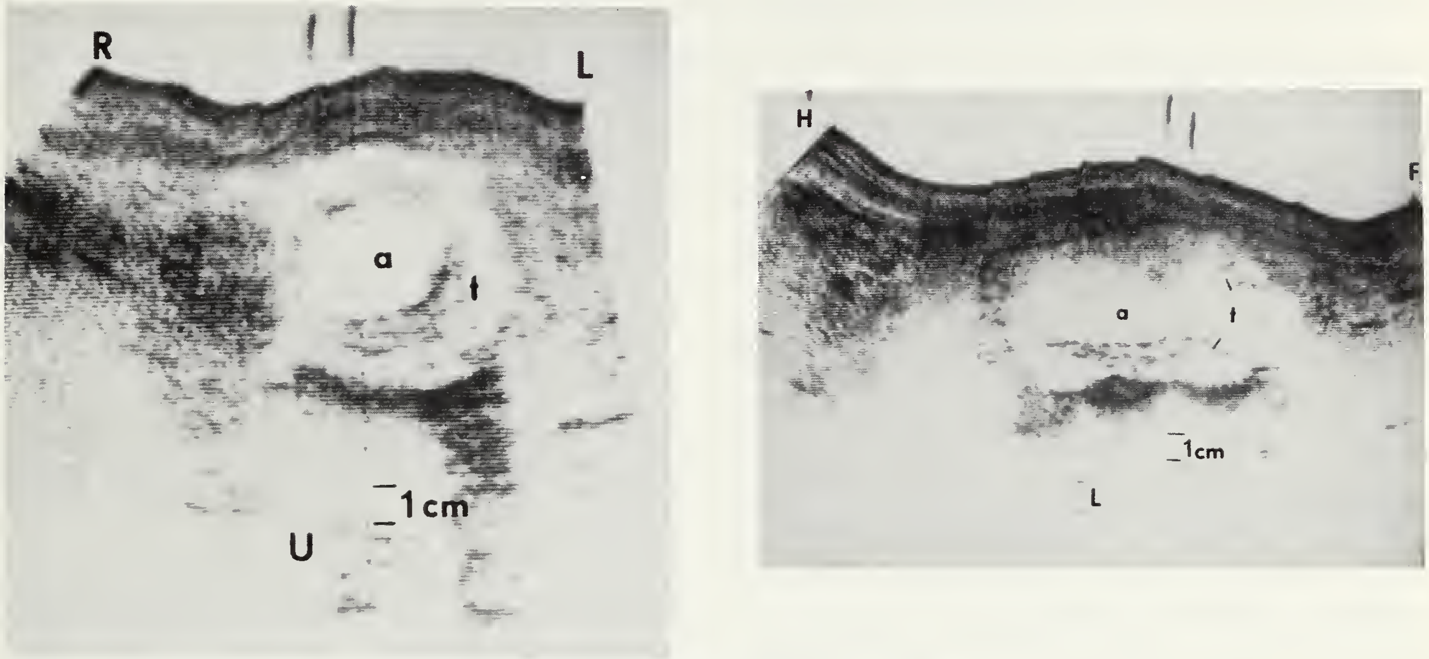

FIGURE 2. 69 year old male with pulsatile abdominal mass.

a. Transverse scan at the umbilicus shows dilatation of the aorta (A) with a diameter of greater than 6 cm Lamellar mural thrombus (T).

b. Longitudinal scan shows the length of the aneurysm, the aortic lumen (A) and extensive thrombus (T).

studies should be done to exclude abdominal aortic aneurysms.

Aortography has been used as a screening procedure but has several drawbacks. Small but definite risks are present due to the use of a catheter and contrast material. Some patient discomfort is almost always present. Both from the complexity of the procedure and the need for hospitalization, total cost is relatively high. Extensive mural thrombus, frequently present, may narrow the lumen to a near normal diameter. While it does not protect the patient from rupture, it may obscure the presence of an aneurysm on aortogram.

Ultrasound has rapidly gained popularity and has been shown to be very accurate in the evaluation of AAA.[1, 2, 3] The aorta is visible by ultrasound in the vast majority of normal patients and never exceeds 3cm in diameter. Ultrasound allows for very accurate measurements to be made (+ or − 3mm) so that the dimensions of the aorta can be recorded free of magnification. Mural thrombus does not interfere with ultrasound so that the aortic lumen, mural thrombus and total size can be measured (Fig. 2A,B). When a small aneurysm is discovered, follow-up examination is easy to perform to check for enlargement. Ultrasound examination is painless and rapidly performed as an outpatient. There are no known risks and total cost is small.

Ultrasound is not totally free of drawbacks, most important of which is the inability of ultrasound to image through gas. Segments of aorta beneath gas filled large or small bowel may not be visible. Usually a repeat study following intestinal cleansing is all that is required to demonstrate the aorta. Stenotic lesions are less well seen than are pathologically dilated ones and narrowing of the aorta or its branches are usually not visible by

FIGURE 3

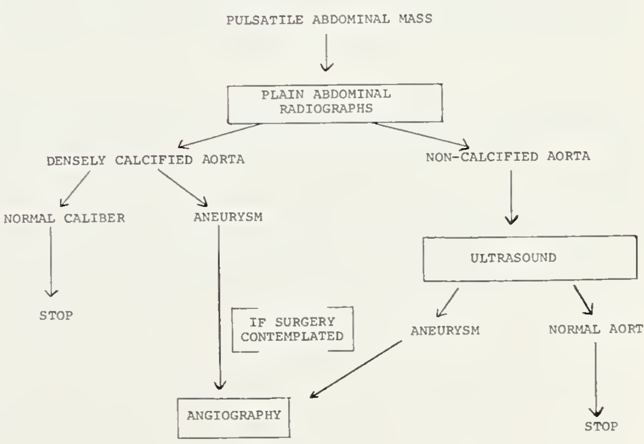

FIGURE 3. A suggested outline for the evaluation of a patient with a pulsatile abdominal mass.

ultrasound. These and peripheral vascular disease are best imaged by contrast angiography. Renal artery involvement by abdominal aortic aneurysm is uncommon.[2] Occasionally the renal arteries will be visible by ultrasound but usually are not. Since renal involvement may alter the management, aortography should be performed prior to surgery.

In conclusion, when a pulsatile abdominal mass is discovered, plain film of the abdomen should be obtained in search for a calcified abdominal aortic aneurysm or calcified normal caliber aorta. If the aorta is not densely calcified, ultrasound should be obtained. If the aorta is aneurysmal and felt to be surgically correctable, angiography should be performed. If the aorta is normal by ultrasound, no further evaluation for aneurysm is necessary.

ACKNOWLEDGEMENTS

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As with all anticholinergics, inhibition of lactation may occur.

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established.

Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated; avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered: isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, *i.e.*, dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

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FAILURE OF CIMETIDINE IN TREATING GIANT DUODENAL ULCER

JOHN L. ORCHARD, M.D.*
WAYNE W. PETERNEL, M.D.**

Most duodenal ulcers are less than one centimeter in diameter.[1] The term giant duodenal ulcer was first applied by Knutsson to those ulcers measuring greater than 2.5 centimeters in diameter radiographically.[2] Giant duodenal ulcers are now being recognized with increasing frequency. Most series of giant duodenal ulcer reported in the literature have advised early operation because of the high incidence of complications, i.e., penetration, hemorrhage, and obstruction.[3, 4, 5]

Cimetidine is superior to placebo in accelerating healing and relieving the symptoms of duodenal ulcer.[6] We treated a patient with a giant duodenal ulcer with cimetidine instead of early operation because he was a poor operative risk. The patient improved clinically but died later from perforation of the giant ulcer while receiving cimetidine. This case is reported as a caveat to physicians who may manage patients with ulcers this large.

CASE REPORT

A 69-year old man first developed pre-hepatic coma ten years prior to admission due to decompensated Laennec's cirrhosis. He discontinued his heavy use of alcohol but had three episodes of upper gastrointestinal bleeding over the next ten years. A bleeding duodenal ulcer was treated medically in 1970. In 1972 he had vagotomy and pyloroplasty for bleeding due to a recurrent penetrating duodenal ulcer. He was treated medically for a third hemorrhage due to duodenal ulcer in 1975.

The patient was admitted December 17, 1976 for treatment of ascites and epigastric pain. He was alert and oriented. Many spider telangiectasia and palmar erythema were present. The murmurs of aortic stenosis and aortic insufficiency were present from chronic rheumatic heart disease. The abdomen was protruberant and a fluid wave was present. The liver span was 16 centimeters and the edge felt 2-3 centimeters below the right costal margin. Moderate pedal edema was present. The stool was guaiac positive.

The admission hemoglobin was 6.7 gm/dl and hematocrit 20%. White count and differential were normal. Liver function tests were as follows: bilirubin total 0.85 mg/dl and 0.3 mg/dl direct, normal SGOT and SGPT, minimally elevated alkaline phosphatase, and albumin 1.8 gm/dl. Prothrombin time was three seconds prolonged.

The patient's ascites was treated with bedrest and diuretics. His epigastric pain was treated with Maalox, 30 milliliters every two hours while awake. On December 26, he had coffee ground emesis and the nasogastric aspirate contained bright red blood. An upper gastrointestinal series showed a deformed duodenal bulb with a large ulcer crater in the roof of the bulb (Figures 1 and 2). A giant ulcer in the roof of the bulb measuring 2.5 × 3 centimeters was seen at endoscopy on January 11, 1977. In addition, the mucosa of the distal one third of the esophagus was friable, erythematous, and covered with a

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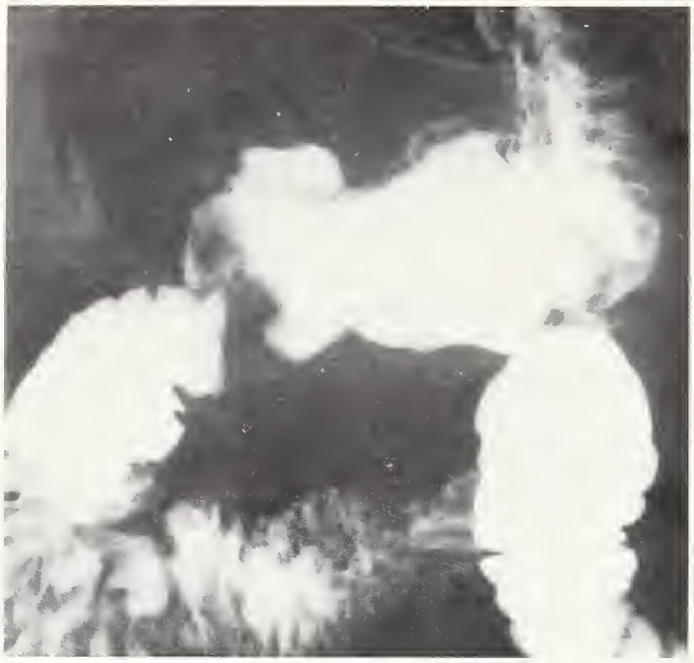

FIGURE 1. Upper Gastrointestinal Series Demonstrates Giant Ulcer in the Duodenal Bulb. Previous Pyloroplasty Obliterated the Radiographic Features of the Pylorus.

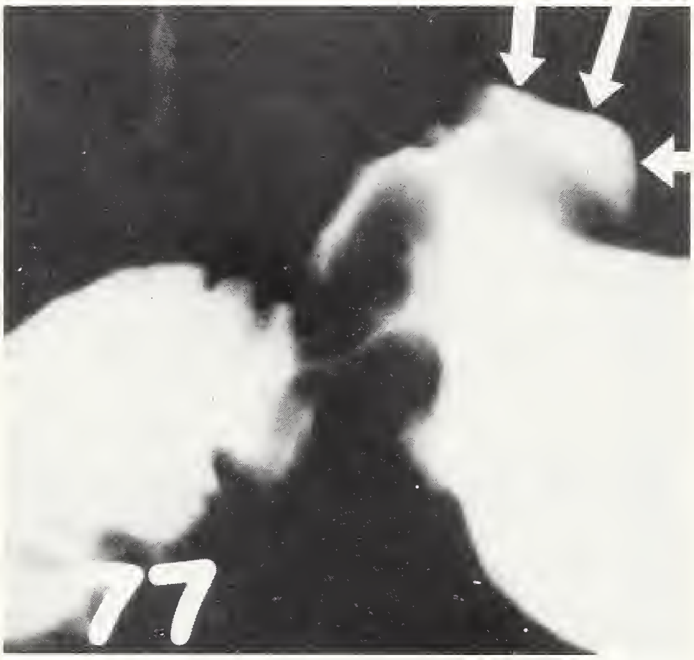

FIGURE 2. Spot Film From the Upper Gastrointestinal Series. Arrows Indicate Giant Duodenal Ulcer.

whitish exudate consistent with moderately severe esophagitis. A one centimeter gastric ulcer was present on the lesser curvature of the antrum. No esophageal varices were seen.

The basal acid output was 2 meq/hr and the maximal acid output was 12 meq/hr.

Surgeons refused to operate because of his high operative risk, therefore, after written consent was obtained, 300 mg of intravenous cimetidine every six hours was begun on January 18, 1977. He was treated with antacids until this time. Within thirty-six hours the patient was pain free. Repeat endoscopy on January 26, showed no endoscopic evidence of esophagitis and complete healing of the gastric ulcer but the giant duodenal ulcer was unchanged. The patient continued to do well clinically until February 7, when he suddenly became hypotensive and developed a fever of 103°. There was no abdominal pain. He was treated with antibiotics for sepsis but died after several hours.

Acute, generalized peritonitis and perforation of the giant duodenal ulcer were found at postmortem examination. The ulcer was in the posterolateral aspect of the duodenal bulb and measured 3 centimeters in diameter. No tumor was present. Ascitic fluid grew many Klebsiella pneumoniae. There was no gross evidence of esophagitis or gastric ulcer. The liver showed micronodular cirrhosis.

DISCUSSION

Since first described by Brdiczka in 1931[7] an increasing number of giant duodenal ulcers have been reported. Review of the literature shows that these patients frequently have a long history of peptic ulcer symptoms such as epigastric pain.[3, 4] Paradoxically, roentgenographic diagnosis of giant duodenal ulcer may be difficult because the barium collection in the ulcer crater may be so large that it is misinterpreted as a normal duodenal bulb.[3, 4, 5] Radiographic narrowing of the duodenal lumen just distal to the ulcer was noted frequently in one series.[5] Early diagnosis is essential because these ulcers are associated with an unusually high incidence of life threatening complications.[3, 4, 5]

Pathologically most giant duodenal ulcers occur in the bulb and have already penetrated into the pancreas at the time of diagnosis.[3, 5] Penetration into the pancreas was present in all ten cases reported by Mistilis.[4]

Most authors advise early operation because of the high incidence of complications such as hemorrhage and obstruction.[3, 4, 5] Very few giant ulcers heal with medical therapy[4, 5] and Kirsh and Brendon feel the mere presence of a giant ulcer should be regarded as an indication for surgery.

Cimetidine reduces all forms of gastric acid secretion by competitively blocking histamine (H-2) receptors. It is superior to placebo in accelerating healing and relieving symptoms of duodenal ulcer.[6] Cimetidine has been used successfully to treat certain cases of the Zollinger-Ellison syndrome.[9]

There are no controlled, double blind studies which compare any form of medical or surgical treatment with placebo to heal giant duodenal ulcer. Although there is no well documented basis for advocating any form of therapy for these patients, review of the literature would indicate that early surgical treatment is preferred to long term medical therapy. While no conclusions can be drawn, we offer the case reported herein as a caveat to physicians until more data on the use of cimetidine to treat giant duodenal ulcer are reported.

SUMMARY

A patient with a giant duodenal ulcer treated with cimetidine is reported. The patient improved clinically but died suddenly from perforation of the ulcer. This case is reported as a caveat to physicians who treat patients with duodenal ulcer. Selected features of giant duodenal ulcer are reviewed. ☐

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ONE HUNDRED THIRTY-FIRST ANNUAL MEETING

The 131st Annual Meeting of the South Carolina Medical Association will be held April 26-29, 1979 in Myrtle Beach, South Carolina. The Myrtle Beach Hilton will again be the site of the various meetings scheduled during that time.

The SCMA House of Delegates will meet on Friday and again on Sunday. Present plans call for meetings of Council on Thursday, April 26, and a scientific program scheduled for Saturday, April 28. Concurrent meetings will be held by the S. C. Pediatric Society, the S. C. Dermatological Society, the S. C. Society of Radiologists and the S. C. Society of Emergency Room Physicians.

New this year will be a formal Presidential Inauguration and Special Awards Presentation, followed by a reception and dancing, to be held late Saturday afternoon. New also will be one registration fee which will include all functions except the SOCPAC Luncheon and will admit all badged attendees and spouses. Fees have been set at $80.00 for members and $120.00 for non-members.

Max Cole, M.D., member of the AMA Board of Trustees from Texas, has accepted an invitation to represent the AMA and address the Council and House of Delegates.

Congressman Butler Derrick has been invited to speak at the SOCPAC Luncheon.

The April, 1979 issue of *The Journal* will contain those committee reports, Resolutions, and Reports of Officers and Councilors which are available at publication deadline. Additional reports received after deadline will be included in the Delegates' Handbooks.

For pre-registration, complete and return the form appearing on page 135 to the SCMA Headquarters office.

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A PHYSICIAN'S USE OF MEDICAL JUDGMENT IN A MALPRACTICE CASE

PAT WARK, B.S.N., M.N., M.B.A.*

One of the issues which frequently arises in a malpractice case is as follows. The patient-plantiff alleges the physician-defendant was negligent in the care he or she provided. The physician responds that, in his opinion, the treatment provided was indicated and appropriate. How does the court handle this conflict? This paper will attempt to answer this question. However, a brief overview of the analytical framework the court uses will make the answer to this question more enlightening.

The determination of whether negligence is involved in a specific act or omission of an act by a physician requires examining three sequential, independent steps:

> "(1) He must possess the degree of professional learning, skill and ability which others similarly situated ordinarily possess;
> (2) He must exercise reasonable care and diligence in the application of his knowledge and skill to the patient's case; and
> (3) He must use his best judgment in the treatment and care of his patient.
>
> If he fails in any one particular, and such failure is the proximate cause of injury and damage, he is liable."[1]

I. *Degree of Learning, Skill and Ability*

Step one in this analysis focuses on the degree of learning, skill and ability possessed by the physician. This level is usually set by statute. State Laws establish and regulate Boards of Medical Examiners which test and license physicians. The level of skill, learning and ability, then, are the minimum ones required to practice in a state. When a physician accepts a patient, he represents that he possesses the ordinary training and skill possessed by similar practicing physicians.

II. *Application of Knowledge, Skill and Ability*

The second step in this analysis focuses on the application of this knowledge, skill and ability. The courts have used two different standards in scrutinizing the application of knowledge, skill and ability. Traditionally, the courts have looked at the practice of similar practitioners in the general neighborhood or in a similar locality. The alleged negligent act would be compared to what other physicians similarly situated would have done. Generalists would be compared to generalists; specialists would be compared to specialists. A rural physician's standard of care could also be different than an urban physician's standard.

The recent trend is to apply a national standard rather than a local one. The defendant-physician's action or non-action in issue is judged in light of what a physician of similar expertise in

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any part of the United States would do — not according to what his local associates would do. There is no South Carolina state court which has addressed this issue. However, the South Carolina Federal District Court[2] endorsed the local community standard. Note the case was decided in 1968 and the issue has not been raised subsequently.

The court also looks to see whether a reasonable level of care, skill and knowledge was exercised. Excellence in this area is not required. The standard used in "the reasonable average merit among ordinarily good physicians."[3] The court does not look at all known physicians and average their care to determine what is average care. Rather, the court looks at what the average physician would do.

III. *Medical Judgment*

The final step in this analysis is to examine the exercise of medical judgment in the treatment and care of a patient. While this is a separate and final step in the analysis, it is considered in the light of a reasonable standard of care. The court generally looks at medical judgment as exercised in the diagnosis of a patient and then in the treatment of the patient.

A. *Diagnosis*

The physician must gather enough data to make a reasonably accurate diagnosis. In a North Carolina case,[4] a psychiatrist continued electroshock treatment on a patient who complained of severe back pains after the first treatment. The treatments were indicated for the patient's emotional disorder, but the clinical symptom of back pain after a treatment should have put the physician on notice that a spinal fracture was a possibility. No x-rays were done and the treatments were continued. The electric voltage for the treatments was also intensified. The court found negligence in the diagnosis because no x-rays were taken when symptoms indicated the injury could involve a fracture. The standard of care used by the court to evaluate this lack of action was the "Standards of Electroshock Treatment" prepared by the Committee on Therapy and approved by the Council of American Psychiatric Association.

The fact that the x-rays which have been taken are negative does not alleviate the physician from further data collection. In one case,[5] the original cervical spine x-rays were negative. However, the patient continued to complain and later it was found that there were multiple fractures of the cervical spine. This court stated it was the physician's duty to use all available diagnostic aids. These aids should be used and re-used if clinical data negates or conflicts with the diagnostic data. Here, expert testimony disclosed that x-rays do not always show fractures and clinical evidence of a fracture may indicate an x-ray should be repeated.

If the physician can document that no basis existed for another diagnosis, he will not be held liable. In a Georgia case,[6] the plaintiff sued the defendant ophthalmologist for damages resulting from a failure to diagnose an intracular penetrating wound caused by a fragment of steel penetrating the cornea and lens of the right eye. The first diagnosis was a corneal abrasion. The ophthalmologist testified that his initial examination did not reveal any evidence which would lead him to suspect a penetrating wound. The court responded that the physician is not responsible for lack of success or honest mistakes or errors in judgment.

The court looks closely at the information gathered by the physician to arrive at his diagnosis. This information comes from the laboratory, the x-ray department and other diagnostic studies as well as from the clinical signs and symptoms manifested by the patient. The facts of a California case[7] revealed a 19 year old boy suffered a head injury and fractured skull in an automobile accident. The physician monitored the boy's pulse, respirations and blood pressure and his pupils' reaction to light. The boy had a steady down-hill course neurologically and died two days after the accident. The autopsy revealed the boy had a brain hemmorhage. The plaintiff claimed the physician was negligent by reason of his failure to diagnose at the outset the hemorrhage and the need for surgery to remove the hematoma. The defendant-doctor contended that the clinical signs of the patient were not classical signs of a hemorrhage and that conservative, non-surgical treatment was recommended and followed by the medical profession for this type of case.

The court responded that ". . . the record presents at most a case of mistaken diagnosis . . ." and, "mere error of judgment, in the absence of a want of reasonable care and skill in the application of his medical learning to the case presented, will not render a doctor responsible for untoward consequences in the treatment of his patient." The collection and evaluation of data should be continuous throughout the entire treatment of a patient. The plan of care should also be modified as the patient's condition changes.

There are no known South Carolina state cases which address the issue of the use of medical judgment. However, the South Carolina Federal District Court addressed this issue.[8] The case involved an eleven year old boy who developed abdominal pain and vomitting. The child was seen by a physician at the dispensary and referred to the emergency room at a nearby hospital after the dispensary physician had run some lab tests. He diagnosed the boy as having appendicitis.

The emergency room physician disagreed with this diagnosis but did not substantiate his diagnosis with physical findings and/or lab and x-ray studies. The boy was seen in the emergency room three times before he was admitted to the hospital. Surgery was performed and it was found that he had a ruptured appendix with a large amount of pus in the abdomen. The delay in performing surgery was said to be the cause of adhesions. The boy, at trial, easily tired and had experienced a personality change.

The court stated that a wrong diagnosis is not malpractice but a physician must use ordinary care in reaching his diagnosis. The court further stated that for liability to attach for a mistake or error in judgment, there must be a failure to comply with a recognized standard of medical care which would be exercised by similar physicians under similar circumstances. The specific acts in issue were: (1) consultation was not sought by the emergency room physician; (2) the clinical signs indicated immediate surgery; and (3) the child should have remained in the hospital for observation.

The court used guidelines set up by the Joint Commission of Hospital Accreditation regarding consultation to conclude that consultation was appropriate in this situation and that a duty was breached when consultation was not sought. The court also found that failure to perform further lab tests or to observe the patient for a certain period of time breached good medical practice. Because of these two decisions the court did not feel it necessary to determine whether surgery was immediately needed.[9] The court said, "Only if a patient is adequately examined is there no liability for an erroneous diagnosis." Therefore the physician must gather sufficient data to make his diagnosis and, if sufficient data have been collected, he is not liable for malpractice if the diagnosis is incorrect. The data in this situation which should have been collected were lab and x-ray studies as well as physical findings.

The court also stated the standard of care is increased if one physician contradicts the diagnosis of another physician. "If competent and reputable physicians have previously made a positive diagnosis that directly conflicts with that of the physician in question, a greater degree of diligence as certain as circumstances and surrounding conditions permit, is imposed on the physician in question in making his diagnosis."[10]

B. *Treatment*

Once the court determines sufficient data are gathered and a proper diagnosis is made, then its attention shifts to whether the treatment given by the physician to the patient was adequate. The court is not willing to substitute its judgment in the proper choice of treatment for that of the physician.

In a Tennessee case,[11] the plaintiff was given radiation therapy for cancer of the cervix of the uterus. The cancer recurred about one and one-half years after the therapy and her physician again advised radiation therapy. Ms. Blankenship's dose was increased the second time. She suffered a third degree burn of her abdomen as a result of these treatments and she developed an ulcer on her abdomen which did not respond to treatment. The ulcer never completely healed.

The expert testimony conflicted. Ms. Blankenship's expert witness stated it was more important to prevent third degree skin burns because this reaction is worse than the cancer. The physician's expert witness stated that the skin burns can always be treated with plastic surgery and the cancer is fatal. Therefore, it is more important to kill the cancer and risk a severe skin reaction.

The court announced the rule required of a physician in this instance:

> "The physicians and surgeons must be allowed a wide range in the exercise of their judgment and discretion. The Science of Medicine is not an exact science. In many instances there can be no fixed rule by

> which to determine the duty of a physician, but he must often use his own best judgment and act accordingly. By reason of that fact the law will not hold a physician guilty of negligence . . ., even though his judgment may prove erroneous in a given case, unless it be shown that the course pursued was clearly against the course recognized as correct by the profession generally. As long as there is room for an honest difference of opinion among competent physicians, a physician who uses his own best judgment cannot be convicted of negligence, even though it may afterward develop that he was mistaken."[12]

Whether an alternate method of treatment can be used is only the initial consideration in a physician's use of his medical judgment. The court also examines whether this treatment was effective and, if not, whether the physician informed the patient of this fact. In a Florida case[13] the physician treated a lip cancer with injections. The lip cancer gradually spread to the patient's chin. The physician discharged the patient in his worsened condition. The expert testimony was in conflict on whether the treatment used was proper or not. Due to the conflict among these physicians, the court ruled no malpractice was involved.

On rehearing, however, the court further determined that the physician had a duty — which he breached — to inform the patient at the earliest possible time that the treatment initially chosen was not effective and that another treatment was needed.

The court, then, puts a constraint on the freedom of judgment given a physician. The physician may choose from among several acceptable treatments and is not liable if his treatment fails. However, if his treatment does fail, he has a duty to inform the patient of this fact and to inform the patient of other available treatments. He must also help the patient secure another type of treatment if his physician is unwilling to change treatments.

A North Carolina case[14] involved a proper diagnosis but an improper treatment. The defendant-pediatrician diagnosed a nine month old child as having asthmatic bronchitis. Part of the treatment prescribed was injections of Strep-Combiotic. The label on the drug had in red capital letters, "NOT FOR PEDIATRIC USE". The amount of drug given to the child was excessive for a child her size and age and as a result of the administration of the drug, she is totally and permanently deaf.

The court said the physician does not insure the success of his treatment or that it will be free of adverse consequences. However, it continued by stating that a pediatric specialist had the duty to exercise reasonable care not only in his diagnosis of the infant's disease, but also in ascertaining probable effects of the drug he prescribes and to observe appropriate precautions in its use. The court felt there may be liability even though other physicians in the locality were using this drug in the same manner the physician-defendant used it.

In summary, the courts examine medical judgment at two levels — diagnosis and treatment. If the physician does not gather enough data, he may be liable in malpractice for a wrong diagnosis. The physician cannot use good medical judgment if he does not have enough information upon which to form an opinion. If the physician gathers adequate information and reasonably updates this information, he will generally not be liable for a mistake in his diagnosis.

Once a diagnosis is made and it is determined not to be a negligent one, the court looks at the treatment provided. The treatment must be acceptable to the medical community. The physician is not liable in malpractice if this treatment fails. Some authority, however, indicates the physician has a duty to try another treatment if his present therapy is not effective or to refer his patient to another physician who is willing to try another type of therapy. ☐

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EUTHANASIA: AN HISTORICAL PERSPECTIVE

JOHN P. DOLAN, Ph.D.*

Although the case of Karen Quinlan must be considered as an important milestone in the current debate on the legal aspects of euthanasia, the more recent decision of the Massachusetts Supreme Judicial Court of November 29, 1977, in the case of Joseph Saikewicz (Mass. Adv. Sh. 2461) which in effect removes from the physician the opportunity of making life and death decisions, makes it clear that as in most bioethical issues, a great deal of ambivalence remains. Unlike the Quinlan case, in which the court approved the decision to disconnect the respirator because, "this should be accepted by a society, the overwhelming majority of whose numbers would, we think, in similar circumstances exercise such a choice in the same way for themselves or for those closest to them," the Massachusetts decision does not require the probate court to make use of the views of attending physicians, ethics committees, or other medical experts. That the decision has opened a hornets' nest can be seen by a sampling of letters to the editor in the *New England Journal of Medicine* and other medical journals throughout the country in recent months.[1]

Since many of the opponents of state interference in the matter of life and death have not infrequently described such proposals as being barbarian and archaic and have laced their anathemas with invectives that recall both the crudities and the refinements of an earlier age, it might be well to follow them literally and see what the ancients as well as more primitive societies have believed and practiced in the matter of euthanasia.

Among uncivilized and aboriginal peoples there is definite evidence that it was common practice to allow the sickly and the elderly to die unaided and in some cases to actually hasten their demise. The English historian S. Hearn informs us that among the Indians of North America at least one half of the aged persons of both sexes who are no longer capable of walking are left alone to starve and perish of want.[2] The Gallinemero tribes in California, as described by S. Powers were wont to kill the elderly when they were no longer able to creep to the forest and collect food and fuel and had become a burden to their offspring. "The poor old wretch is not infrequently thrown on his back and while a stick is placed across his throat two of them sit themselves on it until he ceases to breath."[3] George Catlin, in writing of the tribes of the Great Plains, remarks that the infirm old among themselves uniformly insist upon being left behind claiming that they are no longer of any use — that they left their fathers in the same way — that they wish to die and their children should not mourn them.[4] James Buchanan, early in the nineteenth century, wrote: "Suicide is not considered by the Indians either as an act of heroism or of cowardice, nor is it with them a subject of praise or blame. They view this desperate act as the consequence of mental derangement and the person who destroys himself is an object of pity."[5]

Among the Eskimos of Greenland it was a common practice, when a person was seriously ill, that he consent, if his relatives so requested, to end his sufferings by casting himself into the sea. Special hovels were contructed for the aged so that they could remove themselves from the community and await death in complete isolation.[6] In the Fiji Islands it was regarded as a sign of filial affection to put an aged parent to death and the execution was carried out with all the trappings of a religious ceremony.[7]

Curiously, in the world of classical antiquity, there was, as in our own day, a lack of unanimity on the question of euthanasia. If there is however one distinguishing characteristic that differentiates the outlook of the modern from that of the ancient world it is the element of meliorism that is so much a part of our technically oriented society, the belief that longevity is both a desirable and possible goal for all. This is of course in striking contrast to the mentality of the Hellenistic world which held to the more apologistic view that the prolongation of life of the seriously ill or aged was neither possible nor always desirable.

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Euthanasia was tacitly accepted by many segments of Greek society. While the ancient laws of Thebes and Athens frowned on suicide (shown by the denial of burial rites, confiscation of property, and other signs of degradation), in practice exceptions were permitted, for example, for the seriously ill, or for a soldier not wanting to fall into the hands of the enemy or for a soldier in disgrace. The geographer Strabo tells of the practice on the island of Cos (the birthplace of Hippocrates) in which old people would gather annually for a banquet and together drink a lethal poison.[8] It is said that at Massilia, modern Marseilles, one of the oldest Greek colonies, a death potion was made abailable in a public depository for the use of any citizen who could justify before the Senate the motive for using it — e.g., illness, sorrow, or disgrace.

Abandoning defective or illegitimate offspring seems to have been widely practiced. The absolutist and eugenic control exercised in Sparta is reflected in Plutarch's words,

> The father had no authority to rear his child, when born, but brought it to a place called Lesche; here the elders of his tribe sat and examined the infant. If it were well-made and strong, they bade him rear it, and apportioned to it one of the 9000 allotments of land; but if it were feeble and ill-shaped, they sent it to the so-called Place of Casting Out (Attobetes) — a chasm near Mt. Taygetos — considering that for a child ill-suited from birth for health and vigor to live was disadvantageous alike for itself and for the State.[9]

Greek philosophy, for the most part, held to a person's right to decide his own fate and therefore accepted euthanasia. Plato, for example in the *Laws* follows a restrained path by stating that those who take their lives because of sloth or lack of manliness should be buried alone in such places as are uncultivated and that no column should mark the spot where they are interred.[10] Yet in the same work he fails to censure anyone who by violence deprives himself of his appointed share of life under the compulsion of some painful and inevitable misfortune or out of irremedial and intolerable shame.[11] Writing of infanticide and the care of the deformed newborn, he states: "Those of the inferior parents and any children of the rest that art born defective will be put away in some appropriate manner that must be kept secret."[12] Although he appears at times to have some scruples about suicide, he doesn't hesitate to portray Socrates as a hero for drinking hemlock, and he vigorously opposes attempts to prolong life, saying that medicine is not meant for one whose life is worth nothing to himself and to anyone else.[13]

Aristotle, on the other hand, considered man as owing a duty to the state and thus not justified in taking his own life, a view that was to influence Christian thought in later centuries.[14] As for Aristotle's own death, it is now generally accepted that theories of his taking his own life resulted either from a mistaken attribution to him of the details of the Socrates story of what Anton Chroust calls 'encomiastic biographies' anxious to portray a hero dying a martyr for his own philosophy.[15]

For the ancient Stoics, self-inflicted death was considered morally neutral and at times death at one's own hand was even desirable. Diogenes Laertius writing of Zeno, the founder of Stoicism says ". . . the wise man will for reasonable cause make his own exit from life on his country's behalf, or for the sake of his friends, or if he suffers intolerable pain, mutilation, or incurable disease."[16]

Seneca, the tutor of the young Nero comes close to some contemporary thought when he writes,

> If one death is accompanied by torture, and the other is simple and easy, why not snatch the latter? Just as I shall select my ship when I am about to go on a voyage, or my house when I propose to take a residence, so shall I choose my death when I am about to depart from life. Moreover, just as a long-drawn-out life does not necessarily mean a better one, so a long-drawn-out death does not necessarily mean a worse one. There is no occasion when the soul should be humored more than at the moment of death. Let the soul depart as it feels itself impelled to go; whether it seeks the sword, or the halter, or some draught that attacks the veins, let it proceed and burst the bonds of slavery. Everyone ought to make his life acceptable

> to others besides himself, but his death to himself alone. The best death is the one we like.[17]

Pliny the Younger, who viewed the right to take one's life as God's best gift to men among the sufferings of life,[18] praises his friend Titus Aristo, afflicted by a 'longa et pertinax valetudo' who decides with the consultation of physicians and friends, to determine his own fate.[19] We see the same philosophy in the writings of Epicurus who states: "So death, the most terrifying of ills, is nothing to us, since so long as we exist, death is not with us; but when death comes, then we do not exist. It does not then concern either of the living or the dead, since for the former it is not, and the latter are no more."[20]

It must be recalled that many Roman physicians were actually enslaved Greek prisoners of war at the service of their masters and hence expected to carry out their orders including assisted suicide. The result was that suicide became extremely widespread in Imperial Rome. There were two notable exceptions, the case of the military because of bad example and that of criminals where suicide was regarded as an admission of guilt.[21] Virgil regards those who take their own lives as being unfortunate rather than guilty of crime, regarding them as children who have died prematurely or those condemned to death on false charges.[22]

The advent of Christianity radically changed the attitude of the pagan world towards euthanasia. St. Augustine argued quite categorically that there is no place in the canonical scripture that permits one to destroy himself, either for purposes of attaining immortality or avoiding calamity.[23] This position was entrenched and strengthened by means of early synodal legislation. The Second Council of Orleans in 533, stipulated that the offerings of those who were killed in the commission of any crime may be received except for those who "lay violent hands on themselves."[24] The Council of Braga in 561, excluded from Christian burial those who had committed suicide, a prohibition that was to remain in effect in Canon Law until the present century.[25]

Thomas Aquinas finds that suicide is unlawful for a triad of reasons. In the first place it is contrary to nature as well as to charity; secondly, in killing oneself a person does a disservice to the community to which he belongs; and thirdly because life is a gift of God who alone has the power to kill and to enliven.[26]

The Renaissance witnessed a revival of and interest in the classical world and with it a reexamination of attitudes toward euthanasia. Thomas More, a man canonized by his Church and regarded along with his friend Erasmus as the great spokesman of Christian Humanism appears to condone the taking of one's life under certain conditions in the idealistic society of his famous Utopia:

> They care for the sick with great affection, as I have mentioned, and omit nothing by which these can be restored to health, either by way of medicine or special diet. They console the incurably ill by sitting and talking with them and by alleviating whatever pain they can. Should life become unbearable for these incurables, the magistrates and priests do not hesitate to prescribe euthanasia. Since the priests are looked upon as the interpreters of God, this is considered a pious and holy deed. When the sick have been persuaded of this, they end their lives willingly either by starvation or drugs that dissolve their lives without any sensation of death. Still, the Utopians do not do away with anyone without his permission, nor lessen any of their duties to him. They are convinced that in acting in this manner, they have acquitted themselves honorably. But anyone who inflicts death upon himself when his cause is not approved by the priests and the Senate, is considered worthy neither of the earth nor fire, but is cast unburied into some stinking marsh.[27]

One of the most well known advocates for the legalization of voluntary euthanasia in this country is Walter W. Sackett, a practicing physician elected to the Florida legislature. The main thrust of his argument is that the Federal Constitution be amended to assure citizens "life, liberty, the pursuit of happiness, and the *right to die.*" While Dr. Sackett does not openly favor voluntary euthanasia, yet he argues that a doctor is not morally or legally bound to use extaordinary measures to sustain life, unless a competent patient, or, in the case of an incompetent patient, a responsible relative or guardian requests such measures be used. One of Sackett's most cogent arguments is that unless there is legislation on the matter doctors who refrain from using extraordinary efforts to delay certain death and who use only ordinary means to preserve life may be held liable legally.

Those opposed to this position argue that there is no law that says that a physician must use all the means at his disposal to prolong life, that there is the possibility that legislation of voluntary euthanasia and finally that such an enactment would create problems where none now exist and would make doctors more vulnerable than they are at present to legal suits.

Perhaps one of the more balanced statements on euthanasia and its legal remifications is "The Guidelines Concerning Euthanasia" of the Swiss Academy of Medical Sciences which was recently published for the first time in English by the *New York State Journal of Medicine*. Although is is not to be construed as an official statement of the Medical Society of New York State, we cite it here as a good example of a conciliatory "via media" in this perplexing problem.

> The physician is dependent on the will of the discerning patient who has been duly informed of his illness, its treatment, and the risks involved. Since the discerning patient has to decide himself if he is willing to be treated or not, he has the right to interrupt treatment at any time. Under these circumstances there is no legal basis for the application of therapeutic measures that the patient does not want any more. In this case, in accordance with the desire of the patient, the physician may restrict himself to the administration of drugs or other therapeutic measures that only alleviate suffering. He does not risk being made legally responsible for it. The principle, "voluntas aegroti suprema lex esto" is valid.[28]

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SOUTH CAROLINA MEDICAL ASSOCIATION

AUXILIARY

COMMUNITY HEALTH

The SCMA Auxiliary is greatly concerned with improving the health of South Carolina's families and communities. The Community Health-Family Health Committee serves as the tool for carrying out the health goals of the state organization.

The Auxiliary is supporting the AMA in its national Immunization Awareness campaign. The AMA has set a goal of October 1, 1979, to have 90% of the nation's children fully immunized against childhood diseases. To help accomplish this goal, the county Community Health Committees are continuing to paint "immunization hopscotch" courts on school playgrounds and sidewalks. This year attractive posters, "Don't Let the Bugs Bite You," are being distributed to every elementary and middle school in the state. Hopefully, the posters will emphasize to children and their parents the need for immunization.

Each county Auxiliary has been encouraged to work with local community groups and to plan one specific activity for Senior Citizens. Some projects being undertaken are a Senior Citizens' Day, a tea, a coffee, monthly birthday parties, showing of the film on breast self-examination, needlework classes, Bible study, and book reports. The Auxiliary is concerned with the mental and physical well-being of the aging citizens of South Carolina.

The Richland County Auxiliary was instrumental in acquainting the public with the "Heimlich Maneuver" for saving the lives of choking victims. They printed the instructions on area milk cartons for one month. Several other Auxiliaries are interested in publicizing this procedure.

Health education is a main goal of the Community Health Committee. Numerous Auxiliaries have sponsored and taught courses on Cardio-Pulmonary Resuscitation to their family members and to the public. Programs are underway in the schools to educate children and youth about the problems and dangers of smoking, alcohol and drug abuse. One example is the "Smoking Sam" project of the Greenville County Auxiliary. The Anderson County Auxiliary is using an excellent teaching kit from the American Cancer Society, "An Early Start to Good Health," for school education.

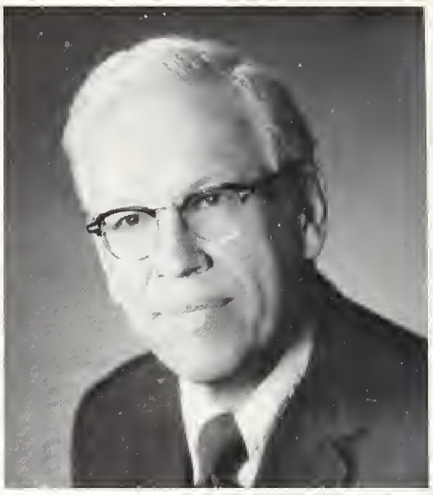
Throughout the state, Auxiliaries are involved with specific needs in their specific communities. These areas include hypertensive screenings, blood donor programs, hospital pediatric clinics, Mental Health Centers, and the Reach to Recovery programs.

Each Auxiliary has been given a set of the packaged programs furnished by the national AMA Auxiliary. In these, tips are given on how to solve major health problems in a community. The Auxiliaries are also encouraged to present programs to effectively deal with the mental, physical, and social good health of its members.

With member involvement and interest, the Community Health Committee will continue to work to improve the quality of life for all South Carolinians.

MRS. JOANNE J. MCCOY, *Chairman*

President's Pages



HOBSON'S CHOICE

This month I would really prefer to write *only* about something pleasant and hopefully productive — the SCMA trip to visit our S. C. Congressional Delegation in Washington, January 31. It was our first organized effort in this regard, and we learned from the AMA Washington office that we happened to be the first state medical delegation to make such a visit this year. However, about twenty states undertake such programs annually, many of them on a much larger scale than ours.

Our SCMA delegation consisted of Doctors Halsted Stone, Harrison Peeples, Waitus Tanner, Bill Hunter, Randy Smoak and John Hawk; and staff members Charlie Johnson and Frank Rogers. We had a worthwhile briefing on legislative issues at the AMA office during the morning, before going to the Hill for a luncheon in the Senate Building arranged through Senator Hollings' office. We are particularly indebted to Ms. Betty Calloway of his staff for making the excellent arrangements. The entire Congressional Delegation, consisting of Senators Thurmond and Hollings, and Representatives Davis, Spence, Derrick, Campbell, Holland and Jenrette, honored us with their presence. Several Congressional staff members, as well as members of the AMA staff, were also present.

We had intended that the luncheon be primarily a social affair, unless the Congressmen desired otherwise. Fritz Hollings soon changed that! His opening gambit was to ask me for my (our) solution to inflation, both general and in the health field. Both Senators and each Representative spoke briefly but to the point on several items of concern. We had a rather lively discussion throughout the luncheon hour.

In the afternoon, members of the SCMA group, accompanied in some cases by the appropriate AMA liaison staff member, met with individual Congressmen for open and frank discussions of health problems in their particular areas.

I am glad that we were able to arrange this Washington meeting *early* in the first sessions of the 96th Congress. We hope that it will enable the SCMA to have more effective input with our representatives in Congress. It should be made an annual affair.

But now, I must move to another topic — one which I have approached in the past with mixed feelings. At present, however, I feel I must face it head on; I must fish or cut bait. The topic — health systems agencies and what we in the medical profession should do about them. Some of you may know all about HSAs; to others they may be just one more three-letter enigma. I won't try to give you an explanation of what they are and how they are supposed to function in South Carolina. For those who really wish to be informed, I suggest you start with reading an article in the S. C. DHEC publication, *Preventive Medicine Quarterly*, Fall, 1978, page four, entitled "Health Planning in South Carolina: Health Systems Agencies," by Linda S. Sugimoto and Louise C. Davenport.

You will learn that under Public Law 93-641, there are four major HSAs in South Carolina; and in addition, Aiken County, because of its location in a Standard Metropolitan Statistical Area, is in a fifth HSA, together with 22 Georgia counties. Hopefully, this latter situation can be resolved under new regulations, and Aiken can re-join South Carolina in one of its HSAs.

You will learn also how HSAs are organized, the composition of their governing Board, how they are supposed to function, the major influence of staff, the potential input of public or corporate members, and many other things.

But to get real insight into HSAs, you must also attend a meeting or two of an HSA Board or one of its committees. It will be an eye-opener to you.

Now we come to Hobson's Choice: what should the medical profession do about HSAs? It appears obvious that without effective advice from the medical profession, the HSA is likely to fail miserably. So there is a real temptation to regard it as a plague, and to say "a pox upon you — stew in your own juice." If we did just that, the HSAs would undoubtedly fail — eventually fall flat on their faces. But in the process, they might do immeasurable and irreparable harm to the public, our patients, and to the medical profession itself.

The alternative, and I am finally convinced that this is what we must do, is for the medical profession — as individuals and as organized societies — to provide the maximum input possible in these consumer dominated organizations.

How can and should this be done? This was discussed in detail at the February 4 conference sponsored by the SCMA at Richland Memorial Hospital entitled, "Relationships — Organized Medicine and Health Planning Agencies." The featured speaker was Dr. William J. Hagood, Jr., President of the Virginia Medical Society and organizer of their Vanguard Program, who delineated their program to "get a handle" on planning agencies. The Vanguard Program has also been portrayed in an article in the October, 1978 issue of *Legal Aspects of Medical Practice.*

The conferees also heard from the Executive Directors of the four South Carolina HSAs, from several physicians who have been active on HSA Boards or on the Statewide Health Coordinating Council, and from the Chairman of a county medical society committee organized to monitor the area HSA. The SCMA Committee on HSAs has been asked to bring to Council specific recommendations for a course of action in this important field. In the meantime, there was general consensus at the Conference on the following:

1. *Physicians and their spouses should be urged to become "public" or "corporate" members of the appropriate HSAs.*
2. *Physicians who so desire and who will and can give adequate time and effort should seek membership on the HSA Board or on important HSA committees in their area.*
3. *County Medical Societies, especially the larger ones, should have broad-based committees representing major specialties to monitor the activities of HSAs and particularly to review project applications and make recommendations to the HSA.*

The Congress, in setting up these agencies under the National Health Planning and Resources Development Act of 1974, clearly established that the so-called "consumers" would have a major voice in health planning. It is time now for the medical profession to assert at every level that physician input is essential and must be heeded as well as heard for such planning to be rational and effective, rather than counter-productive or even destructive.

John C. Hawk, Jr., M.D.
President

A reminder

ZYLOPRIM®

(allopurinol)

100 and 300 mg scored Tablets

- inhibits uric acid formation
- helps prevent urate crystal depositions in synovia
- reduces risk of uric acid lithiasis

INDICATIONS AND USE: This is not an innocuous drug and strict attention should be given to the indications for its use. Pending further investigation, its use in other hyperuricemic states is not indicated at this time.

Zyloprim® (allopurinol) is intended for:

1. treatment of gout, either primary, or secondary to the hyperuricemia associated with blood dyscrasias and their therapy;
2. treatment of primary or secondary uric acid nephropathy, with or without accompanying symptoms of gout;
3. treatment of patients with recurrent uric acid stone formation;
4. prophylactic treatment to prevent tissue urate deposition, renal calculi, or uric acid nephropathy in patients with leukemias, lymphomas and malignancies who are receiving cancer chemotherapy with its resultant elevating effect on serum uric acid levels.

CONTRAINDICATIONS: Use in children with the exception of those with hyperuricemia secondary to malignancy. The drug should not be employed in nursing mothers.

Patients who have developed a severe reaction to Zyloprim should not be restarted on the drug.

WARNINGS: ZYLOPRIM SHOULD BE DISCONTINUED AT THE FIRST APPEARANCE OF SKIN RASH OR ANY SIGN OF ADVERSE REACTION. In some instances a skin rash may be followed by more severe hypersensitivity reactions such as exfoliative, urticarial and purpuric lesions as well as Stevens-Johnson syndrome (erythema multiforme) and very rarely a generalized vasculitis which may lead to irreversible hepatotoxicity and death.

A few cases of reversible clinical hepatotoxicity have been noted and in some patients asymptomatic rises in serum alkaline phosphatase or serum transaminase have been observed. Accordingly, periodic liver function tests should be performed during the early stages of therapy, particularly in patients with pre-existing liver disease.

Patients should be alerted to the need for due precautions when engaging in activities where alertness is mandatory.

Nevertheless, iron salts should not be given simultaneously with Zyloprim. This drug should not be administered to immediate relatives of patients with idiopathic hemochromatosis.

In patients receiving Purinethol® (mercaptopurine) or Imuran® (azathioprine), the concomitant administration of 300-600 mg of Zyloprim per day will require a reduction in dose to approximately one-third to one-fourth of the usual dose of mercaptopurine or azathioprine. Subsequent adjustment of doses of Purinethol or Imuran should be made on the basis of therapeutic response and any toxic effects.

Usage in Pregnancy and Women of Childbearing Age: Zyloprim® (allopurinol) should be used in pregnant women or women of childbearing age only if the potential benefits to the patient are weighed against the possible risk to the fetus.

PRECAUTIONS: Some investigators have reported an increase in acute attacks of gout during the early stages of allopurinol administration, even when normal or subnormal serum uric acid levels have been attained.

It has been reported that allopurinol prolongs the half-life of the anticoagulant, dicumarol. This interaction should be kept in mind when allopurinol is given to patients already on anticoagulant therapy, and the coagulation time should be reassessed.

A fluid intake sufficient to yield a daily urinary output of at least 2 liters and the maintenance of a neutral or, preferably, slightly alkaline urine are desirable to (1) avoid the theoretic possibility of formation of xanthine calculi under the influence of Zyloprim therapy and (2) help prevent renal precipitation of urates in patients receiving concomitant uricosuric agents.

Patients with impaired renal function require less drug and should be carefully observed during the early stages of Zyloprim administration and the drug withdrawn if increased abnormalities in renal function appear.

In patients with severely impaired renal function, or decreased urate clearance, the half-life of oxipurinol in the plasma is greatly prolonged. Therefore, a dose of 100 mg per day or 300 mg twice a week, or perhaps less, may be sufficient to maintain adequate xanthine oxidase inhibition to reduce serum urate levels. Such patients should be treated with the lowest effective dose, in order to minimize side effects.

Mild reticulocytosis has appeared in some patients.

As with all new agents, periodic determination of liver and kidney function and complete blood counts should be performed especially during the first few months of therapy.

ADVERSE REACTIONS:

Dermatologic: Because in some instances skin rash has been followed by severe hypersensitivity reactions, it is recommended that therapy be discontinued at the first sign of rash or other adverse reaction (see WARNINGS). Skin rash, usually maculopapular, is the adverse reaction most commonly reported.
Exfoliative, urticarial and purpuric lesions, Stevens-Johnson syndrome (erythema multiforme) and toxic epidermal necrolysis have also been reported.
A few cases of alopecia with and without accompanying dermatitis have been reported.
In some patients with a rash, restarting Zyloprim (allopurinol) therapy at lower doses has been accomplished without untoward incident.

Gastrointestinal: Nausea, vomiting, diarrhea, and intermittent abdominal pain have been reported.

Vascular: There have been rare instances of a generalized hypersensitivity vasculitis or necrotizing angiitis which have led to irreversible hepatotoxicity and death.

Hematopoietic: Agranulocytosis, anemia, aplastic anemia, bone marrow depression, leukopenia, pancytopenia and thrombocytopenia have been reported in patients, most of whom received concomitant drugs with potential for causing these reactions. Zyloprim® (allopurinol) has been neither implicated nor excluded as a cause of these reactions.

Neurologic: There have been a few reports of peripheral neuritis occurring while patients were taking Zyloprim. Drowsiness has also been reported in a few patients.

Ophthalmic: There have been a few reports of cataracts found in patients receiving Zyloprim. It is not known if the cataracts predated the Zyloprim therapy. "Toxic" cataracts were reported in one patient who also received an anti-inflammatory agent; again, the time of onset is unknown. In a group of patients followed by Gutman and Yü for up to five years on Zyloprim therapy, no evidence of ophthalmologic effect attributable to Zyloprim was reported.

Drug Idiosyncrasy: Symptoms suggestive of drug idiosyncrasy have been reported in a few patients. This was characterized by fever, chills, leukopenia or leukocytosis, eosinophilia, arthralgias, skin rash, pruritus, nausea and vomiting.

OVERDOSAGE: Massive overdosing, or acute poisoning, by Zyloprim has not been reported.

HOW SUPPLIED: 100 mg (white) scored tablets, bottles of 100 and 1000; 300 mg (peach) scored tablets, bottles of 30, 100 and 500. Unit dose packs for each strength also available.

Complete information available from your local B. W. Co. Representative or from Professional Services Department PML.

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Editorials

JIM CLARK

JAMES ROLAND CLARK, M.D.
June 29, 1937 — February 2, 1979

To his community he was Dr. James R. Clark, a good doctor and a good citizen. To us he was Jim, a shy and self-effacing man who was in many ways a model physician.

He practiced high quality internal medicine in a quiet, efficient way. At a given time, he was likely to have had more acutely ill hospitalized patients under his care than any other Columbia physician. He took on many responsibilities, some of them known to but a few persons. He was especially adept at dealing with the physical and psychological ravages of sickle cell disease.

The same morning that Jim, his wife Yvonne, his seven-year-old daughter Ynola, and his mother-in-law were slain, he was to have met with two young residents to discuss the possibility of a special sickle cell disease clinic for indigent patients.

A native of Columbia, Jim was valedictorian and student council president at C. A. Johnson High School. He served as president of both the student council and his senior class at Blackburn College in Carlinville, Illinois, and then did graduate work in biochemistry at Washington University in St. Louis. After graduating with honors from Howard University Medical School, he interned at the Wayne County General Hospital in Detroit, spent two years as captain in the Army Medical Corps, and completed his training in internal medicine at the University of California Medical Center in San Francisco. While in San Francisco, he volunteered his services at a free clinic in the Haight-Asbury district.

Jim served as chairman of the Task Force on Hypertension of the South Carolina Heart Association, and was a founding member and medical advisor of the Columbia Area Sickle Cell Anemia Foundation.

Jim Clark was a passionate, concerned physician. He sought consultations for his patients not only from other physicians, but also from social workers, dietitians, and physical therapists, who recall the unusual extent to which he specified exactly *how* he desired their assistance. His attendance at hospital committee meetings was outstanding, and his name was seldom if ever to be found on the long "delinquent" lists of those with uncompleted hospital charts. In short, he set an excellent example.

The only criticism we ever heard made of Jim Clark was the one by his secretary after his death: "He was probably too good."

— CSB

LEGIONNAIRES' DISEASE IN SOUTH CAROLINA — AN UPDATE

Legionella pneumophila is the proposed name for Legionnaires' disease bacterium. Sophisticated analytical processes were used to determine that this organism is genetically unrelated to all other organisms tested at both the genus and species level. Hence, a new genus and new specific epithet for a previously undescribed species.

Although Legionnaires' disease occurs world-wide, the most intensive studies have been from the United States and Great Britain. In the United States, neither sporadic cases nor epidemics of this disease have shown any particular geographic distribution.

Legionnaires' disease was first recognized as a unique entity in 1976. Subsequent retrospective studies of samples stored from undiagnosed outbreaks indicate that the disease and the etiological agent have been present for many years. For example, the studies have shown that there were 81 cases in an outbreak in a Washington, D. C. hospital in 1965 and 144 cases from a health department in Michigan in 1968.

Searching deeper into the freezer at the Center for Disease Control (CDC), produced two isolates obtained in 1947 from patients with undiagnosed septicemia which have now been identified as *L. pneumophila*. These identifications are further evidence that Legionnaires' disease is not new, but rather newly recognized.

It appears that Legionnaires' disease is seasonal with all outbreaks occurring between May and November. It is estimated that 2-3% of undiagnosed pneumonia is Legionnaires' disease and that there are seven subclinical cases for each clinical case.

Legionnaires' disease most frequently presents as a unilateral or bilateral pneumonia with symptoms of fever ($\geq$ 39°C), malaise, myalgia and cough. As more information is obtained from studying sporadic cases, a wider spectrum of signs and symptoms appear to be associated with this infection. Nausea, vomiting and diarrhea without pneumonia may be the presenting symptoms. Delirium, confusion and disorientation have occurred as have hallucinations and grand mal seizures. Renal insufficiency, hyponatremia ($<$ 130 mM/1), abnormal liver function tests and disseminated intravascular coagulation are frequently described.

Additional findings which support a diagnosis of Legionnaires' disease as opposed to other types of pneumonia are: 5-7 day history of "viral pneumonia", cough with little or no sputum, lymphopenia ($< 1000/\text{mm}^3$), WBC $< 15{,}000/\text{mm}^3$, hematuria, encephalopathy, elevated SGOT and hypoalbuminemia. Radiological evidence of progression of pneumonia from an interstitial infiltrate to alveolar consolidation and clinical deterioration in the face of antibiotic therapy also support the diagnosis.

The efficacy of erythromycin as the drug of choice is not universally accepted. However, sensitivity to erythromycin is supported by *in vitro* studies and this antibiotic has been used by most investigators. Parenteral administration is recommended in the beginning, with a transition to oral therapy as the patient responds to medication. Two grams of erythromycin should be administered daily for three weeks. The addition of rifampin to the therepeutic regimen has given a good clinical response for some physicians.

Immunosuppression, cigarette smoking, preexisting chronic illness and heavy alcohol consumption appear to influence the susceptibility to this disease. The sequelae of Legionnaires' disease are: (1) patient is confused for several months, (2) X-ray evidence of disease is slow to resolve (4-8 months) and (3) antibodies persist for about two years.

Gross pathology usually reveals an increased lung weight. The cut surface of the lung is moist and airless, usually being described as congested, but not hemorrhagic. Distribution of the lesions is diffuse with no obvious segmental distribution. Confluent broncho-pneumonia is frequent and discrete nodules or lesions with well demarcated borders are infrequent. Abscesses are sometimes seen. Pleuritis occurs in some cases with a layer of fibrin over the pleura.

Conventional staining procedures are not suitable for *L. pneumophila*. It cannot be seen in H and E stained sections and it is very difficult to see by the Brown and Brenn stain. The best visualization is obtained by the Dieterle silver impregnation procedure. Some investigators prefer a modification of the Giemsa stain because this will reveal the organisms and the tissue reaction. The direct fluorescent antibody stain may also be used on tissue sections. The organisms are usually found within polymorphonuclear neutrophiles and macrophages.

Most of the inflammatory reaction is in the alveoli and not the bronchi and scarring may completely obliterate the lung tissue. Histologically there is little evidence of acute pneumonia. An organizing pneumonia with interstitial inflammation and fibrosis is seen. Focal necrosis of the pulmonary parenchyma and desquamation of alveolar epithelium may also be present, as may septal inflitration with lymphocytes and macrophages and alveolar consolidation by neutrophils, histiocytes, red cells and fibrin. There are no granulomatous reactions. Capillary and epithelial basement membranes are usually intact.

The indirect fluorescent antibody (IFA) procedure is presently being used to measure antibody to *L. pneumophila*. Antibody usually takes three weeks to appear, so it is necessary to submit multiple serum specimens. One sample should be collected at the onset of illness, another sample three weeks later and a third sample six weeks after onset. A single titer of ≥ 1:256 or a four-fold rise between two specimens to 1:128 is considered diagnostic.

The prevalence of antibody in the general population and the persistence of antibody after infection are not known with certainty. It has been shown that 54% of the Legionnaires in Philadelphia who were not ill at the time of the outbreak had antibody. Whether this was due to a previous infection or a sub-clinical infection cannot be stated. Antibody persisted for at least two years in 94% of the 31 survivors of the Philadelphia outbreak. Based on small studies, prevalence has been estimated to be 2-5% in the "normal" population.

L. pneumophila is a fastidious, gram negative rod, 0.3 to 0.9U by 2U. It may elongate to a filamentous form in culture but does not exceed 8U in human lung tissue. The ends may be blunt or pointed. The organism is weakly oxidase positive, catalase positive, starch positive, liquefies gelation and produces a soluble brown pigment. It does not hydrolyze urea, reduce nitrates or ferment carbohydrates. The special media used to isolate *L. pneumophila* must be incubated under CO_2 at 35°C. In three to four days the growth is sparse, with good growth in seven to eight days.

Specimens that should be submitted for culture are lung biopsy and pleural fluid. Sputum, trans-tracheal aspirates and bronchial washings have rarely been a good source of the organism.

It has recently been shown that there are four distinct serogroups with no cross staining between serogroups. Fortunately, 86% of the human isolates to date are in serogroup 1, the only serogroup for which diagnostic reagents are presently available. Serogroup 1 reagents are being used at the S. C. Department of Health and Environmental Control Bureau of Laboratories.

Although there are many sporadic cases, most outbreaks of this disease have occurred in hospitals and hotels where the individuals were probably exposed to a common source. Although there is no evidence of person to person spread, one study of hospital personnel has shown an increased incidence of antibody among respiratory therapy personnel. Construction sites and air conditioning systems have consistently been associated with outbreaks. The extensive procedure necessary to recover *L. Pneumophila* from nature has limited the number of isolations of the agent and thus the ecological niche of the organism has yet to be established.

There have been four isolates of the organism from nature. Three were from cooling towers in buildings associated with outbreaks and one was from a stream close to where an outbreak occurred. Laboratory evidence has demonstrated that *L. pneumophila* will survive in water for as long as one year, but that it will not persist in properly treated cooling tower water for more than 48 hours.

From these data, it is suggested that there is airborne transmission of *L. pneumophila* and the reservoirs are probably water and soil. The incubation period of the illness is apparently two to ten days.

Several new tests are being developed to aid the clinician in making a diagnosis. To increase specificity, polyvalent conjugates are being developed to identify all serogroups of *L. pneumophila* by direct staining of tissue or four serological tests. There are four main thrusts in developing aids to an earlier diagnosis: new serological tests are under study (ELISA, microagglutination, hemagglutination); a chemically defined medium for isolation of the organism is being formulated; attempts are being made to detect *L. pneumophila* antigen in urine; and studies are being conducted to detect exotoxin from the organism.

To aid you in establishing a diagnosis of Legionnaires' disease, the Bureau of Laboratories can offer the following services:.

1. culture of lung biopsy material or pleural fluid (trans-tracheal aspirates are less satisfactory),
2. serological testing for antibody titer,
3. direct fluorescent antibody staining of tissue.

Seven cases of Legionnaires' disease have been diagnosed by this laboratory, Six positive specimens were identified from 48 paired sera submitted. From an additional 41 single serum specimens, one case had a significant titer. The patients came from Charleston, Greenville, Lancaster, Richland (2), Anderson and Chester counties. Five patients were male and two were females. Based on the limited data available, we have estimated the onset of disease in these patients to be February (1), March (1), August (1), September (3), October (1), thus conforming with the seasonal pattern defined by data from other sources.

The Bureau of Laboratories is ready to assist in establishing the incidence and prevalence of this disease in South Carolina and in aiding in the individual physician with diagnostic tools.

Arthur F. DiSalvo, M.D., Chief
Bureau of Laboratories
South Carolina Department of Health and Environmental Control
Columbia, S. C. 29201

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Brief Summary

INDICATION: Tenuate and Tenuate Dospan are indicated in the management of exogenous obesity as a short-term adjunct (a few weeks) in a regimen of weight reduction based on caloric restriction. The limited usefulness of agents of this class should be measured against possible, risk factors inherent in their use such as those described below.

CONTRAINDICATIONS: Advanced arteriosclerosis, hyperthyroidism, known hypersensitivity, or idiosyncrasy to the sympathomimetic amines, glaucoma. Agitated states. Patients with a history of drug abuse. During or within 14 days following the administration of monoamine oxidase inhibitors, (hypertensive crises may result).

WARNINGS: If tolerance develops, the recommended dose should not be exceeded in an attempt to increase the effect; rather, the drug should be discontinued. Tenuate may impair the ability of the patient to engage in potentially hazardous activities such as operating machinery or driving a motor vehicle; the patient should therefore be cautioned accordingly. *Drug Dependence*: Tenuate has some chemical and pharmacologic similarities to the amphetamines and other related stimulant drugs that have been extensively abused. There have been reports of subjects becoming psychologically dependent on diethylpropion. The possibility of abuse should be kept in mind when evaluating the desirability of including a drug as part of a weight reduction program. Abuse of amphetamines and related drugs may be associated with varying degrees of psychologic dependence and social dysfunction which, in the case of certain drugs, may be severe. There are reports of patients who have increased the dosage to many times that recommended. Abrupt cessation following prolonged high dosage administration results in extreme fatigue and mental depression; changes are also noted on the sleep EEG. Manifestations of chronic intoxication with anorectic drugs include severe dermatoses, marked insomnia, irritability, hyperactivity, and personality changes. The most severe manifestation of chronic intoxications is psychosis, often clinically indistinguishable from schizophrenia. *Use in Pregnancy*: Although rat and human reproductive studies have not indicated adverse effects, the use of Tenuate by women who are pregnant or may become pregnant requires that the potential benefits be weighed against the potential risks. *Use in Children*: Tenuate is not recommended for use in children under 12 years of age.

PRECAUTIONS: Caution is to be exercised in prescribing Tenuate for patients with hypertension or with symptomatic cardiovascular disease, including arrhythmias. Tenuate should not be administered to patients with severe hypertension. Insulin requirements in diabetes mellitus may be altered in association with the use of Tenuate and the concomitant dietary regimen. Tenuate may decrease the hypotensive effect of guanethidine. The least amount feasible should be prescribed or dispensed at one time in order to minimize the possibility of overdosage. Reports suggest that Tenuate may increase convulsions in some epileptics. Therefore, epileptics receiving Tenuate should be carefully monitored. Titration of dose or discontinuance of Tenuate may be necessary.

ADVERSE REACTIONS: *Cardiovascular*: Palpitation, tachycardia, elevation of blood pressure, precordial pain, arrhythmia. One published report described T-wave changes in the ECG of a healthy young male after ingestion of diethylpropion hydrochloride. *Central Nervous System*: Overstimulation, nervousness, restlessness, dizziness, jitteriness, insomnia, anxiety, euphoria, depression, dysphoria, tremor, dyskinesia, mydriasis, drowsiness, malaise, headache; rarely psychotic episodes at recommended doses. In a few epileptics an increase in convulsive episodes has been reported. *Gastrointestinal*: Dryness of the mouth, unpleasant taste, nausea, vomiting, abdominal discomfort, diarrhea, constipation, other gastrointestinal disturbances. *Allergic*: Urticaria, rash, ecchymosis, erythema. *Endocrine*: Impotence, changes in libido, gynecomastia, menstrual upset. *Hematopoietic System*: Bone marrow depression, agranulocytosis, leukopenia. *Miscellaneous*: A variety of miscellaneous adverse reactions has been reported by physicians. These include complaints such as dyspnea, hair loss, muscle pain, dysuria, increased sweating, and polyuria.

DOSAGE AND ADMINISTRATION: Tenuate (diethylpropion hydrochloride): One 25 mg. tablet three times daily, one hour before meals, and in midevening if desired to overcome night hunger. Tenuate Dospan (diethylpropion hydrochloride) controlled-release: One 75 mg. tablet daily, swallowed whole, in midmorning. Tenuate is not recommended for use in children under 12 years of age.

OVERDOSAGE: Manifestations of acute overdosage include restlessness, tremor, hyperreflexia, rapid respiration, confusion, assaultiveness, hallucinations, panic states. Fatigue and depression usually follow the central stimulation. Cardiovascular effects include arrhythmias, hypertension or hypotension and circulatory collapse. Gastrointestinal symptoms include nausea, vomiting, diarrhea, and abdominal cramps. Overdose of pharmacologically similar compounds has resulted in fatal poisoning, usually terminating in convulsions and coma. Management of acute Tenuate intoxication is largely symptomatic and includes lavage and sedation with a barbiturate. Experience with hemodialysis or peritoneal dialysis is inadequate to permit recommendation in this regard. Intravenous phentolamine (Regitine®) has been suggested on pharmacologic grounds for possible acute, severe hypertension, if this complicates Tenuate overdosage.

Product Information as of April, 1976

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References: 1. Citations available on request—Medical Research Department, MERRELL RESEARCH CENTER, MERRELL-NATIONAL LABORATORIES, Cincinnati, Ohio 45215. 2. Hoekenga, M.T., O'Dillon, R.H., and Leyland, H.M.: A Comprehensive Review of Diethylpropion Hydrochloride. International Symposium on Central Mechanisms of Anorectic Drugs, Florence, Italy, Jan. 20-21, 1977.

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8-3921 (Y587A)

LETTER TO THE EDITOR

To the Editor:

Having just read the December issue of the Journal of SCMA which features an entire page as an ad for physicians to join SOCPAC — your voice for unified campaign action — it seemed fitting to record a small fact for now and posterity. The last physician to serve in the Congress of the United States from South Carolina was Thomas Jefferson Strait, M.D., 1893-1899. Therefore, since I was a candidate for Congress from the First District in the 7 November 1978 election, I thought all physicians and, especially, SOCPAC would be interested and helpful in my effort. Not so! The American Medical Political Action Committee gave my opponent, Mendel Davis, a total of $4,900.00 on 15 September 1978. I received no contribution from this organization.

C. C. Wannamaker, M.D.
311 N. Montague Avenue
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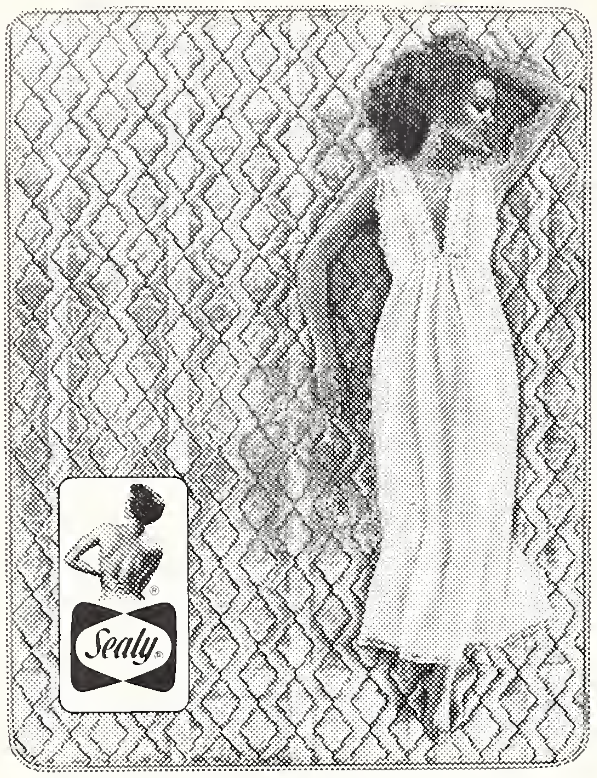

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THE JOURNAL
OF THE SOUTH CAROLINA MEDICAL ASSOCIATION
VOLUME 75 APRIL 1979 NUMBER 4

ONE HUNDRED THIRTY-FIRST ANNUAL MEETING

THE MYRTLE BEACH HILTON
MYRTLE BEACH, S. C.

The 131st Annual Meeting of the South Carolina Medical Association will be held April 26-29, 1979 in Myrtle Beach, South Carolina. The Myrtle Beach Hilton will again be the site of the various meetings scheduled during that time.

The SCMA House of Delegates will meet on Friday and again on Sunday. Present plans call for Council to meet on Thursday, April 26, and the SCMA scientific program is scheduled for Saturday, April 28. Concurrent meetings will be held by the S. C. Pediatric Society, the S. C. Dermatological Society, the S. C. Society of Radiologists and the S. C. Society of Emergency Room Physicians. Available information regarding these meetings is included in this issue.

New this year will be a formal (black tie optional) Presidential Inauguration and Special Awards Presentation, followed by a reception, to be held beginning at 6:00 p.m. on Saturday, April 28. New also will be one registration fee which will include all functions except the SOCPAC Luncheon and will admit all badged attendees and spouses. Fees have been set at $80.00 for members and $120.00 for non-members.

Max Cole, M.D., member of the AMA Board of Trustees from Dallas, Texas, has accepted an invitation to represent the AMA and address the Council and House of Delegates.

Congressman Butler Derrick has been invited to speak at the SOCPAC Luncheon.

Included in this issue of *The Journal* are those committee reports, Resolutions, and Reports of Officers and Councilors which were available at publication deadline. Additional reports received after deadline will be included in the Delegates' Handbooks.

131st SOUTH CAROLINA MEDICAL ASSOCIATION ANNUAL MEETING

THE MYRTLE BEACH HILTON, MYRTLE BEACH, SOUTH CAROLINA
APRIL 26 - APRIL 29, 1979
GENERAL PROGRAM

Thursday, April 26, 1979

8:00 a.m. - 9:00 a.m.	SCMA Council Buffet Breakfast — West Ballroom, Suite C
9:00 a.m. - 12:30 p.m.	SCMA Council Meeting — West Ballroom, Suites A and B
12:30 p.m.	SCMA Council Buffet Luncheon — West Ballroom, Suite C
2:00 p.m. - 5:00 p.m.	SCMA Council Meeting — West Ballroom, Suites A and B
2:00 p.m. - 5:30 p.m.	State Board of Medical Examiners Hearings — Parlours I-IV
3:00 p.m. - 6:00 p.m.	Exhibits Open — Lobby and Mezzanine
3:00 p.m. - 8:00 p.m.	SCMA Registration — Lobby

Friday, April 27, 1979

7:30 a.m. - 5:00 p.m.	SCMA Registration — Lobby
7:30 a.m. - 9:00 a.m.	SCMA Council Buffet Breakfast and Meeting — Parlours II-IV
7:30 a.m. - 9:00 a.m.	SCMA Reference Committee Chairmen Buffet Breakfast and Meeting — Parlour I
9:00 a.m. - 12:00 Noon	State Board of Medical Examiners Board Meeting — Parlour VI
9:00 a.m. - 12:00 Noon	SCMA House of Delegates Meeting — Center and West Ballrooms
9:00 a.m. - 6:00 p.m.	Exhibits Open — Lobby and Mezzanine
12:30 p.m. - 2:00 p.m.	State Board of Medical Examiners Luncheon — Alfredo's Restaurant
12:30 p.m. - 2:30 p.m.	SOCPAC Board Meeting — Parlour VI
1:30 p.m. - 3:00 p.m.	MUSC Alumni Luncheon — Center Ballroom — Honoring Class of 1929
2:00 p.m. - 5:00 p.m.	State Board of Medical Examiners' Workshop — West Ballroom, Suite A
3:00 p.m. - 6:00 p.m.	SCMA Reference Committee Meetings (Will be staggered and room assignments will be given in Delegates' Handbooks) — West Ballroom, Suites B and C; and Parlours I-IV and VI
7:00 p.m.	1939 and 1943 MUSC Reunion Receptions and Dinners — West Ballroom, Suites A, B, C

Saturday, April 28, 1979

7:30 a.m. - 5:00 p.m.	SCMA Registration — Lobby
7:30 a.m. - 9:00 a.m.	SCMA Council Buffet Breakfast and Meeting — West Ballroom, Suite C
9:00 a.m. - 12:00 Noon	State Board of Medical Examiners Committee Meetings — Parlour VI
9:00 a.m. - 1:15 p.m.	SCMA Scientific Session — East Ballroom
1:15 p.m. - 2:30 p.m.	SOCPAC Luncheon — West Ballroom
2:00 p.m. - 5:00 p.m.	S. C. Academy of Family Physicians Board of Directors Meeting — Parlour VI
2:00 p.m.	Exhibits To Be Closed — Lobby and Mezzanine

6:00 p.m. - 7:00 p.m.	SCMA Presidential Inauguration and Special Awards Presentation Ceremony — Center and West Ballrooms
7:00 p.m. - Until	SCMA President's Reception and Dancing — Center, East and West Ballrooms
7:30 p.m.	SCMA Door Prize Drawing — Center, East and West Ballrooms (During SCMA President's Reception and Before Dancing)

Sunday, April 29, 1979

7:30 a.m. - 9:00 a.m.	SCMA Council Buffet Breakfast and Meeting — Parlours I-III
7:30 a.m. - 9:00 a.m.	SCMA Reference Committee Chairmen Buffet Breakfast and Meeting — Parlour IV
7:30 a.m. - 10:30 a.m.	SCMA Registration — Lobby
9:00 a.m. - 12:00 Noon	SCMA House of Delegates Meeting — Center and East Ballrooms
12:00 Noon	Swearing-In Ceremony of All SCMA Officers Except President to be Followed By a Ten-Minute, Non-Denominational Service — Center and East Ballrooms
12:30 p.m.	SCMA Council Reorganization Meeting — Center and East Ballrooms
12:45 p.m.	S. C. Medical Care Foundation Annual Membership Meeting — Parlours I-IV

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SCMA SCIENTIFIC PROGRAM*

MYRTLE BEACH HILTON, MYRTLE BEACH, S. C.
APRIL 28, 1979

ADVANCES IN DIAGNOSTIC MEDICINE

MODERATOR: Joseph C. Ross, M.D., Professor and Chairman
Department of Medicine, M.U.S.C

9:00 - 9:20	Prenatal Diagnostic Technology	*Edgar O. Horger, M.D.* Professor of Obstetrics and Gynecology, M.U.S.C.
9:20 - 9:40	Diagnosis of Learning Difficulty	*Daniel L. Moore, M.D.* Assistant Professor of Pediatrics Spartanburg General Hospital
9:40 - 10:00	Echocardiography in Children	*Ashby B. Taylor, M.D.* Assistant Professor of Pediatrics, M.U.S.C.
10:00 - 10:20	Echocardiography in Adults	*Bruce W. Usher, M.D.* Assistant Professor of Medicine, M.U.S.C.
10:20 - 11:00	Neuroradiology in Diagnosis of Cerebrovascular Disease	*Edward L. Hogan, M.D.* Professor and Chairman Department of Neurology, M.U.S.C. *G. Douglas Hungerford, M.D.* Associate Professor of Radiology, M.U.S.C.
11:00 - 11:20	Coffee Break	
11:20 - 11:40	Radioimmunoassay in Endocrine Diagnosis	*Jon H. Levine, M.D.* Associate Professor of Medicine, M.U.S.C.
11:40 - 12:10	Swan-Ganz Catheter in Cardiovascular Diagnosis	*William P. Hood, Jr., M.D.* Professor of Medicine School of Medicine University of Alabama
12:10 - 12:45	Advances in Diagnostic Oncology	*Harvey M. Golomb, M.D.* Assistant Professor of Medicine Section of Hematology/Oncology Pritzker School of Medicine University of Chicago
12:45 - 1:15	Questions and Discussion	Drs. *Horger, Moore, Taylor Usher, Hogan, Hungerford, Levine, Hood, & Golomb*

* Credit for Category 1 AMA Physicians' Recognition Award and AAFP credits applied for.

ELECTIONS 1979

OFFICERS:	President-Elect* First Vice President Second Vice President Secretary Treasurer (Nominated by Council)
COUNCILORS:	Dist. 1: J. Frank Biggers, M.D. Bartolo M. Barone, M.D. (Both elected in 1978, but must stand for election again in 1979 in order to fulfill the "odd-even" requirement) Dist. 2: James H. Herlong, M.D. (Elected in 1976, term ending in 1979, must stand for re-election in 1979 for what will necessarily be a one-year term in an "even" district) Dist. 3: E. Mims Mobley, M.D. (Elected in 1977, must stand for re-election in 1979 because of "odd-even" requirement) Dist. 5: Halsted M. Stone, M.D.* (Elected 1970; served three full terms; vacancy in this district) Dist. 7: Davis D. Moise, M.D. (Elected in 1975, re-elected in 1978. Must stand for re-election in 1979 on "odd-even" basis) Dist. 8: Randolph D. Smoak, Jr., M.D. (Elected in 1972 to fill unexpired term of Harvey Atwill, M.D. Elected in 1973 to first three-year term. Re-elected in 1976 for second full term. Eligible for re-election in 1979, but will have to be for a one-year term on the "odd-even" basis.) Dist. 9: Euta M. Colvin, M.D. (Elected in 1972 to fill unexpired term of Harold Hope, M.D. Re-elected in 1974 to full three-year term, and re-elected in 1977 for three-year term. Because of "odd-even" requirement must stand for re-election in 1979.)
AMA DELEGATE:	C. Tucker Weston, M.D., term expires 12/31/79
AMA ALTERNATE DELEGATE:	J. D. Gilland, M.D., term expires 12/31/79
SPEAKER OF THE HOUSE:	J. Gavin Appleby, M.D. Elected 1978 to fill unexpired term of Wm. H. Hunter, M.D.
VICE SPEAKER OF THE HOUSE:	Hugh Wells, M.D. Elected 1978 to fill unexpired term of J. Gavin Appleby, M.D.
STATE BOARD OF MEDICAL EXAMINERS:	Second District: Foster Marshall, M.D., term expires Fifth District: C. W. Brice, Jr., M.D., term expires

* Indicates *not* eligible for re-election.

REPORTS OF COUNCILORS

SECOND DISTRICT

I have enjoyed serving you another year as Councilor of the Second District of the South Carolina Medical Association. A tremendous amount of material continues to come before your Council. Meetings have been long and we have tried to put a great deal of thought and effort in the decisions that we have made.

I have served on the Constitution and Bylaws Committee which has been very active this year. As you know we have had the job of finalizing the revision of the Constitution and Bylaws which hopefully will go into effect this year. This is going to incorporate many changes in the structure of the SCMA. We feel that this revision will better serve the membership of the Association. All changes will be fully approved by the House of Delegates.

A major effort has been made this year to serve our members in many different areas of concern — i.e., Legislative matters, Federal and State Regulatory Agencies, revision of the Constitution and Bylaws, a further effort to broaden our professional liability insurance program, continuing medical education projects, increasing our membership, and many more.

I attended the weekend Council Work Retreat last fall and have tried to attend all Council Meetings. I attended a recent joint seminar in Columbia on the role of organized medicine in the HSA's in South Carolina.

I am trying to improve the communication between activities of the SCMA and our local Medical Society members. I will be submitting periodic articles to the *Recorder*, which is the monthly publication of the Columbia Medical Society. I have tried to attend as many meetings as possible of the monthly Executive Committee Meetings of the Columbia Medical Society. I would also encourage everyone to try to read the monthly publication of the SCMA, the *Newsletter*, which always includes brief items of interest to everyone of us and to our daily practice of medicine.

It has indeed been my pleasure to work with the many fine officers of your association. I would also compliment the fine work and support that is given by the staff of the SCMA. I will continue to pledge my support and my efforts to the very best of my ability.

Sincerely,
JAMES H. HERLONG, M.D.

It has been a pleasure to serve as the second Councilor from the Second District during the past year. I have been in attendance at all Council meetings during the year, and have attended one Executive Committee meeting at their request. I have also enjoyed attendance at all of the Aiken County Medical Society meetings.

There have been no significant problems within the Second District which have required my special attention. I look forward to continuing to represent the members of the Second District over the next year.

It has also been my pleasure to serve as the Chairman of the Ad Hoc Committee on Nurse-Midwifery. This committee has not completely finalized its activity, but we will look forward to having a report to present to the House of Delegates at the April meeting.

Respectfully submitted,
KENNETH N. OWENS, M.D.

SIXTH DISTRICT

Having served as Councilor for the Sixth Medical District, I would like to make the following report concerning meetings I have attended and other activities which have occurred within the Sixth Medical District in the past year.

I attended several meetings of the local medical societies, particularly those in Marlboro County, Florence County, Horry County, Dillon County and Marion County. I also had contact with several hospital staff organizations regarding activities and problems within the medical staff.

Doctors Stone and Tanner, along with Mr. Charles Johnson and myself, attended a meeting with the Board of Trustees and Medical Staff of Chesterfield County Hospital concerning problems they had in their activities. I was unable to attend the second meeting which was held; however, we have attempted to assist the Medical Staff and the Board of Trustees in working out problems concerning staff activities at the Chesterfield Hospital. A further report will be coming later on concerning future activities in this area.

Florence County held a reception for the Legislative Delegation from Florence County, including the four Senators from the same Senatorial District. It is felt that some good mileage was obtained from this. The members of the delegation and their wives attended; they seemed to enjoy the relationship of openness with the citizens of Florence County.

Horry County was reorganized. They have adopted a new set of By-Laws, established considerable activity, and have monthly meetings with scientific speakers. I have attended two or three of these meetings and have found them excellent. The Horry County Medical Society has also established a newspaper column concerning medical topics in the county newspaper. This is being done as a courtesy to the newspaper and this column cannot be given to other papers within the Horry County area. Horry County also held a dinner for the members of the Horry County legislative delegation. Following the meeting, a very "hot" discussion was held concerning legislative topics. Considerable input and information was exchanged. This meeting was very well attended and the discussion was very lively.

Frank Rogers and I were invited to attend the Dillon County Medical Society. The meeting was well attended by both physicians and their wives. We attempted to stimulate interest in SCMA and SOCPAC during the meeting.

There have been no particular problems in other counties in the Sixth District, and things seem to be moving quite well. I was glad to attend the Columbia Medical Society Annual Banquet. This is always a highlight of the year, and was enjoyed by all who were present.

JAMES S. GARNER JR., M.D.

NINTH DISTRICT

This has been a good year for The Ninth District. A few problems have arisen but I believe they have been satisfactorily handled.

The Spartanburg County Medical Society has been very active with good participation in SCMA activities. *The Bulletin* published by The Society continues to improve and will, I believe, hold its own with other similar publications in the state. The Spartanburg Society has been honored to have as speakers at two of its meetings — Hoyt Gardner, M.D., President-Elect of The American Medical Association, and The Honorable Richard Riley, Governor of South Carolina.

A number of new physicians have come into our district in the past year and we are trying to get them interested and involved in SCMA activities. As our area continues its industrial development, there will be continued opportunities for additional physicians in Cherokee, Spartanburg and Union Counties.

We have a continuing problem with the chiropractic issue in our area particularly since we now have a chiropractic school in Spartanburg County. This is a problem that is more immediately critical for our district but is one that affects the entire state as well. We must continue to be alert and resist intrusion by these people into the practice of medicine.

Respectfully submitted,
EUTA M. COLVIN, M.D.

COMMITTEE REPORTS

INSURANCE COMMITTEE

Last year as Chairman of the Insurance Committee, I reported to you, the SCMA House of Delegates, that the South Carolina Medical Association had achieved a very major breakthrough dealing directly with the physician disability insurance problem and also with office overhead protection.

In November this disability program along with office overhead, dependent life, property, home owners, etc. was offered to the South Carolina Medical Association members. A letter and an insurance brochure were mailed to every physician member of the SCMA announcing this program. The insurance committee met and examined the various coverages being offered. At the committee's request I wrote to each county medical society president asking them to allow Mr. W. Floyd York of the Professional Insurance Agency, Inc. to be present at one of their meetings to explain the various insurance programs available.

As of the date of this report the response and results have been poor.

Since it is the charge of this committee to examine and recommend to Council insurance programs to serve as benefits for Association members the committee is actively seeking a dental insurance program to offer to the SCMA members and would like to ask for input from the House of Delegates for additional insurance benefits in order to meet the true insurance needs of the South Carolina Medical Association members.

The Insurance Committee has been studying the SCMA Blue Cross/Blue Shield Group Claims Experience Report for the purpose of rate stabilization with hopes of preventing large scale rate increases over the next few years.

I am happy to report that for the first time in several years there has not been a rate increase in the South Carolina Medical Association Blue Cross/Blue Shield Health Insurance Program.

It is the plan of the committee to continue this study in order to maintain the present premium level and provide the South Carolina Medical Association members with the highest quality insurance plans available.

Respectfully submitted,
DAGGETT O. ROYALS, M.D., *Chairman*

ALCOHOLISM AND DRUG ADDICTION

As Chairman of this committee, I have met on several occasions with a special committee constituted to study the abuse of prescription drugs in South Carolina. This committee was composed of other professional organizations and governmental agencies that interface on the drug abuse problem.

During this past year, I have also served, by the appointment of Governor Edwards, on the Task Force to study the laws relating to beer and wine in South Carolina.

S. HUNTER RENTZ, M.D., *Chairman*

THE DISABLED DOCTORS PROGRAM

Contact has been made and I have personally appeared before the State Medical Auxiliary enlisting their support in assisting the Impaired Physicians committee in South Carolina. Dialogue is currently being developed with the SCMA Mental Health Committee to formulate a retreat seminar for marital enrichment and sharing of mutual practice stresses and the impact that they may have on the physician, his marriage and his family.

S. HUNTER RENTZ, M.D.,
Chairman

COMMITTEE ON MEDICAL EDUCATION

The South Carolina Medical Association Committee on Medical Education has been in a re-organization process since the resignation and retirement of former Chairman, Vince Moseley, M.D. During the past year, the new Chairman has attempted to direct the committee's activities in a way which is compatible with the wishes of the physicians in South Carolina with regard to continuing medical education.

I have been assisted in committee activities, particularly with development of the questionnaire, by Gerald E. Costello, Ed.D., an Assistant Professor with the Department of Health and Physical Education at the University of South Carolina, and Mrs. Joy Drennen of the SCMA staff. The committee has welcomed a new member, Benton M. Montgomery, M.D., and accepted with regret the resignations of E. Mims Mobley, M.D., and David C. Askins, M.D.

The committee, of course, has continued its on-going activities in the form of certification and re-certification processes for the awarding of Category 1 credits for the Physicians' Recognition Award of the AMA. By the time this report is published, both Columbia Medical Society and Spartanburg General Hospital will have been considered for re-certification for the awarding of Category 1 AMA Physicians' Recognition Award credits. This year, the re-certification was handled in the form of reverse site visits.

Also, Anderson General Hospital will have completed the requirements for certification, with pre-survey questionnaires and an on-site visit. Hopefully, certification for the awarding of Category 1 credits will have been approved.

During the past year, members of this committee have attended both the AMA Meeting on Continuing Medical Education in Chicago, and the Alliance for Continuing Medical Education meeting in New Orleans. It was felt that input was needed as to what is taking place regarding continuing medical education in other parts of the country, and the reports from these meetings have been very helpful to the committee as a whole in beginning to formulate its program for CME in South Carolina.

Most important to the committee in determining the desires and requirements of physicians in South Carolina as they relate to CME and the role of the SCMA has been the questionnaire on CME which has been mailed to the entire physician population in the state. The return on the questionnaire was very encouraging, and a tabulation of the results will be attached to this report when presented to the House of Delegates and will also be published later in final form in *The Journal.* The results are presently being studied by a special Ad Hoc Committee charged with the task of developing a proposed five-year plan for continuing medical education in South Carolina and what role the SCMA will take regarding this very important matter. We regret that we will not have this plan finalized by the 1979 Annual Meeting of the SCMA for presentation to the House. A target date of July 1, 1979 has been established for finalization of the plan. It was felt the plan is of such importance to the membership and the entire physician population of the state, that it should not be developed prematurely or without full and careful consideration.

We would ask this House of Delegates, however, to endorse and approve the concept of the plan and authorize the SCMA Council to issue final approval.

Lastly, it is felt important at this time to state the present concept of the SCMA Committee on Medical Education which the committee agrees consists of (1) maintaining proper accreditation of those facilities approved for CME credit; (2) assisting in record keeping of CME credits as a service to physicians in the state; and (3) not becoming involved in actual programming of medical meetings in competition with medical schools, hospital staffs, etc., since the committee's function remains in the area of supervising and assisting those institutions already accredited.

I wish to personally thank the committee members for their excellent attendance at our meetings and for their cooperation in the difficult process of reorganization and relocation of offices to the SCMA Headquarters.

Respectfully submitted,
N. B. BAROODY, JR., M.D., *Chairman*

THE SCMA INSURANCE PEER REVIEW COMMITTEE

The SCMA Insurance Peer Review Committee continues to meet regularly on the first Thursday of alternate months.

There have been two major issues in which the committee has been involved over the past year, one of them being policies. We are happy to announce that we have been able to inform Blue Cross and Blue Shield of the problems of practicing physicians in collections as well as procedures that were not covered by the carrier and which are now being covered. We have been able to point out certain problems, such as medical physicians not being paid when their patients end up with surgical diseases. This problem is currently being addressed by Blue Cross and Blue Shield, and I am sure that we will have a satisfactory remedy and solution in the near future.

We have been able to convince Blue Cross and Blue Shield to use physicians in active practice to do their review work for them as consultants. The physicians currently being used come mainly from the S. C. Academy of Family Practice, as well as Internal Medicine physicians in the state. We are pleased to announce that the quality of the review process has improved considerably since the change was made to practicing physician consultants.

We are still concerned about the unfairness of retroactive denials, and are working to find a solution to eliminate this procedure. One of the possibilities being considered at the present time is concurrent review on patients while they are in the hospital rather than a review of records after their discharge. Several hospitals have already signed contracts with Blue Cross and Blue Shield for concurrent reviews, thus eliminating the possibility of retroactive denials.

Our committee still continues to work with the problem of patterns of practice reviews. We feel that this type review has been helpful in improving the quality of medical care, with a decrease in over-utilization of hospital admissions and lengths of stay.

We feel that the committee has been and will continue to be an effective sounding board for Blue Cross and Blue Shield as well as our fellow physicians.

Most recently, the committee has been working with the South Carolina Department of Social Services in an attempt to devise a contract to allow for peer review of Medicaid claims. We are hopeful that we will be able to advise them as we have Blue Cross and Blue Shield for the benefit of the physicians in South Carolina so that they will have a more just review process. We are trying to coordinate Blue Cross and Blue Shield and DSS review processes to save the physicians from two separate review procedures.

We feel that we have an excellent committee. We have seen cooperation from the insurance carriers to implement most of the suggestions that we have made. We feel that we have been helpful in pointing out problems that the carriers were unaware of. We will continue to strive to preserve the quality of medical care in South Carolina.

Respectfully submitted,
DANIEL W. BRAKE, M.D., *Chairman*
ALBERT E. CREMER, M.D., *Vice Chairman*

MEDICAL ASPECTS OF SPORTS

The Committee on the Medical Aspects of Sports has had two (2) conference calls this year and sponsored five (5) sports clinics around the State. Activity has already begun on this year's clinics which we hope to schedule in May and have it mandatory for high school coaches to attend.

The Committee has proposed and Senator Heyward E. McDonald has introduced legislation which will regulate and license athletic trainers in South Carolina. This bill has been patterned after a Georgia law which that state has found to be workable. The Committee urges all SCMA members to support this legislation.

Respectfully Submitted,
EMMETT M. LUNCEFORD, JR., M.D.,
Chairman

COMMITTEE TO THE SOUTH CAROLINA DEPARTMENT OF CORRECTIONS

At its meeting on September 27, 1978, the Committee to the South Carolina Department of Corrections was requested to assist in the program to improve and accreditate medical care and health services in South Carolina jails. To implement the program in South Carolina, a Jail Health Services Program Advisory Committee was formed with Charles F. Crews as its Chairman. The members of the Comm. to the South Carolina Department of Corrections were also appointed to this statewide Advisory Committee. Other members of the Advisory Committee are representatives from a number of county detention facilities throughout the state, as well as representatives from the Governor's Office of Criminal Justice and the South Carolina Department of Corrections.

By way of background with regard to this project, the SCMA and the AMA are cooperating on a program to improve medical care and health services in jails under a Law Enforcement Assistance Administration (LEAA) funded project. This accreditation program is an outgrowth of a 1972 AMA nationwide survey which disclosed a lack of attention given to medical care in the nation's jails. Upon approval of the LEAA grant in November 1975, the AMA proceeded in three major program areas: the development of model health care delivery systems in jails; the development of minimum standards for health care in jails, and the implementation of a national certification program; and the establishment of a clearinghouse for information on jail health care.

In an effort to keep the accreditation program at a management level, only a few states that had expressed interest in being pilot project states were selected to participate in the testing of the standards and accreditation process. South Carolina was selected as one of the pilot project states and received a sub-grant from the AMA in the spring of 1978 to begin the accreditation process.

Utilizing the monies received from the AMA, the SCMA contracted with the consulting firm of Carter.Goble.Roberts, Inc. to provide the staff support necessary for coordinating the project within the state.

In an effort to solicit voluntary participation, descriptive announcements and applications were sent to thirty selected city and county jails throughout the state. Subsequently, eight jails submitted applications for participation in the program. Because of budget and time constraints, only six of the jails were recommended to the AMA for participation in the program by the selection sub-committee. Once the applications from these jails were approved by the AMA, each jail received a self-survey questionnaire which required the medical and other staff to evaluate their procedures on health care delivery in terms of compliance with the AMA-developed standards. While the degree of compliance with the standards varied from jail to jail, all the jails returning the survey questionnaires indicated that they had a physician or medical authority responsible for the jail's medical services. While some of the jails indicated that they did have acceptable medical procedures (based upon the standards), they were usually not written or systematic.

Upon analysis of the self-survey questionnaires, exemplary policies and procedures and recommended medical practices for health care in jails, developed by the AMA, were sent to each jail. It was expected that each jail would use the examples to structure their health care delivery system so as to comply with the standards.

At this time, two of the six jails have, based upon a second survey questionnaire, restructured and/or written the necessary policies and procedures to comply with the minimum number of standards required for accreditation. The next step in the accreditation process for these two jails will be an on-site survey conducted by a survey team consisting of physician and non-physician members. The field report from the on-site survey team, which will review all aspects of jail operations and administration related to medical care, will be forwarded to the AMA National Advisory Committee for final action. It is anticipated that the on-site surveys for these two facilities will be completed by mid-April.

The Program to Improve Medical Care and Health Services in South Carolina's jails appears to be successful. The Jail administrators involved believe that the use of the standards in terms of self-evaluation of their health care systems assists in the identification of shortcomings in their medical practices and aids in the requests to county or city councils for monies to provide health care to inmates. While it is realized that the demand on physician time is very great, there is a substantial segment of the South Carolina population that is confined in jails who need the attention and care of the medical community.

Respectfully submitted,
CHARLES F. CREWS, M.D.,*Chairman*

COMMITTEE ON PUBLIC RELATIONS

On January 25, 1979, the Committee on Public Relations was given this charge:

"The Committee shall be responsible for developing programs and activities which will increase the public's awareness of the role and objectives of the medical profession and of the South Carolina Medical Association in society.

"It shall also consider programs which will provide useful health education information for the public.

"The Committee, working with staff, shall also coordinate the Association's relationship with the news media in South Carolina and shall recommend programs which will further this relationship."

During the past year through meetings and by correspondence, the Committee on Public Relations working in consultation with W. LeRoy Harrelson Associates, Inc. has endeavored to carry out the above charge.

LEON BANOV, JR., M.D., *Chairman*

Librax®
Each capsule contains 5 mg
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Please consult complete prescribing information, a summary of which follows:

Indications: Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

"Possibly" effective: as adjunctive therapy in the treatment of peptic ulcer and in the treatment of the irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis.

Final classification of the less-than-effective indications requires further investigation.

Contraindications: Glaucoma; prostatic hypertrophy, benign bladder neck obstruction; hypersensitivity to chlordiazepoxide HCl and/or clidinium Br.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Physical and psychological dependence rarely reported on recommended doses, but use caution in administering Librium® (chlordiazepoxide HCl) to known addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions) reported following discontinuation of the drug.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.**

As with all anticholinergics, inhibition of lactation may occur.

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established.

Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated; avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered: isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, *i.e.*, dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

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MEDICAL ADVISORY COMMITTEE TO THE DEPARTMENT OF VOCATIONAL REHABILITATION

The South Carolina Medical Association Advisory Committee to the Vocational Rehabilitation Agency met Thursday, February 15, 1979, at the Vocational Rehabilitation Comprehensive Center, West Columbia, South Carolina. Dr. Ben N. Miller, Chairman, presided. Members of the committee present were: Dr. Roy C. Campbell or Orangeburg, Dr. Robert S. Clarke, Jr. of Due West, Dr. Joseph R. Dorchak of Spartanburg, Dr. Lloyd R. MacLean of West Columbia, Dr. Basil Manly, IV of Greenville, Dr. Thomas W. Messervy of Columbia, Dr. Gerald J. Quinn of Charleston, Dr. John R. Sanders of Greenville, Dr. David E. Tribble of Columbia, and Dr. Ted Watson of Greenville. Dr. Braxton B. Wannamaker attended on special invitation by the Agency. (Alec E. Brown, DDS of Columbia represented the Dental Association.)

Vocational Rehabilitation Agency was represented by: Mr. Joe S. Dusenbury, Commissioner; Dr. Robert E. Brabham, Assistant Commissioner — Field and Case Services; Mr. Tommy Hadwin, Supervisor — VR-PS and Youth Services; Mr. Walter J. House, Case Service Audit Supervisor; Mr. Paul Knight, Program Specialist; Mr. T. E. Ringer, Jr., Assistant Commissioner — Administration and Special Services; Mr. Vince Rhodes, Supervisor — Statewide Programs and Facilities; Mr. Raynold Stoudemayer, Supervisor — Case Services Division; Mr. Richard Vandiver, Director — Disability Determination Division; and, Dr. William F. Ward, Jr., Medical Director — Vocational Rehabilitation Comprehensive Center.

Dr. Ben N. Miller recalled how the South Carolina Medical Association had established the Medical Advisory Committee, and how active it has been in providing assistance to Vocational Rehabilitation.

Mr. Joe Dusenbury was presented and he outlined the increase in the medical fees schedule and indicated that the CPT Coding System would be fully implemented by July 1, 1979.

Mr. Paul Knight gave a very informative talk concerning the treatment of cancer clients. He stated that South Carolina Vocational Rehabilitation Agency could become more involved in cancer rehabilitation and that the Agency had written a grant request for monies to fund a study. At present Vocational Rehabilitation has not been involved to a great extent in providing cancer treatment, but it was noted that in 1976 we had served approximately 300 cancer clients, providing other rehabilitation services with success. Research during the last five years has devised new methods for cancer treatment and there is a better prognosis for certain types of cancer. Vocational Rehabilitation wants to take a closer look to see how we can become more involved with the vocational rehabilitation aspects of the cancer patients. It was pointed out that the thing we need most is a better means of selecting cancer patients, since at present we do not have the expertise to decide which patients can be effectively served. The purpose of submitting the grant is to seek funds to develop a screening selection system to indicate which cancer cases would be more acceptable for Vocational Rehabilitation involvement. It was noted that this project will be localized in the Columbia area serving nine counties. It was learned that in 1978 approximately 8,200 new cases of cancer would be diagnosed in South Carolina with approximately 1,925 of these being in the regional area where the study will begin. The nine counties which will make up the pilot project will be (this depends on funding) Lexington, Richland, Kershaw, Fairfield, Lancaster, Newberry, Sumter, Lee and York. The pilot project will be based in the Columbia Area Office utilizing the Richland Memorial Hospital as a referral source. It is hoped that we will hear from this grant request by March, 1979, as to whether it will be accepted for funding. Mr. Knight made it clear that we would not have funds to be heavily involved in sponsorship of cancer treatment.

Dr. William F. Ward, Jr., who serves as Medical Director of the Comprehensive Center gave an informative talk concerning the facility and its function. He stated that he was very proud of the facility and felt that it was something we could continue to see develop in the months and years to come. He commented briefly on the staff which includes Registered Nurses, Licensed Practical Nurses, and assistants in other areas. Local physicians assist when needed. It was pointed out that this is not a facility for acute cases, but a few types of acute conditions are handled as they arise. Dr. Ward cited two accomplishments, one was to see clients being able to do things for themselves that they never thought possible before. The second was that after they had been through a program of evaluation the staff is able to report back to the physician, or local rehabilitation counselor, that the client is now able to be trained to function in some type of employment. It was noted that those patients who were not interested in getting better did not improve in most instances, but there have been some with poor interest who did improve after receiving services through the facility. Dr. Ward expressed his appreciation to the staff of the facility for their hard work and indicated that they played a big part in the rehabilitation of the clients. Again, it was emphasized that this is not an acute medical care facility, but as the facility becomes more sophisticated, possibly it could be expanded to an acute medical center. Appreciation was expressed to the many physicians who have helped provide services to the clients.

The film, "A Different Approach," was shown to the group and all concerned received it very favorably indicating that it was very interesting and informative.

A tour of the facility was conducted by the Vocational Rehabilitation Staff for the committee members. The response from the members was that they were very impressed with the facility and its function in serving the handicapped people of South Carolina.

A question and answer period concerning the facility, and the Agency followed. The committee members responded with a number of questions which were answered by the Vocational Rehabilitation Staff. The committee members were impressed and pleased with the work being done by Vocational Rehabilitation.

Mr. Dusenbury stated to the committee that if they saw anything amiss in the performance of the Agency, they should feel free to contact him, or if they prefer, contact Dr. Miller.

The Chairman thanked the Committee for their attendance and stated that he was well pleased with the attendance as only three members were absent. He also expressed his thanks to the staff members at the facility for the meal and their work in preparing for the meeting. There being no further business or discussion, the conference adjourned.

BEN N. MILLER, M.D., *Chairman*

COMMITTEE ON OCCUPATIONAL MEDICINE

The main functions of this committee are: (1) To serve as liaison between the South Carolina Medical Association and its member physicians and the South Carolina Industrial Commission in that maintaining a healthy dialogue between these two groups is important to the good will and satisfaction of all parties involved in the delivery of appropriate and effective and complete medical care to the workers in the state affected with occupational illness or injury. (2) To serve in an advisory capacity to the Industrial Commission as regards its fee schedule. (3) To serve as an arbitrating force in fee disputes between the Commission and the individual physicians and to discourage over-treatment and improper treatment. (4) To act as a resource for the South Carolina Medical Association in the field of Occupational Medicine. (5) To further education and training in the area of occupational health.

At a meeting in Columbia, August 16, 1978, this committee met with Senator Isadore Lourie who is Chairman of the Senate Study Committee on Workmen's Compensation. Senator Lourie reported the status of current legislation affecting Workmen's Compensation and reviewed an analysis of workmen's compensation laws in South Carolina relating to their degree of compliance with the 1970 passage by Congress of the Occupational Health and Safety Act. Dialogue between this committee and Senator Lourie was both informative and productive. Senator Lourie told the committee that South Carolina was one of the few states which had a committee such as the one he chairs which by state statute is functioning. His committee has a full time staff and funding. There are three study commissions in the South Carolina House, three in the South Carolina Senate and a Governor's study commission with six members which include an attorney representing the claimants, one representative of industry, one of labor, one from the general population, and one from the Governor's office. In regard to compliance of South Carolina with the Occupational and Health Safety Act of 1970, there were twenty-three points considered and it appears that South Carolina is in better shape than many other states in regard to compliance.

The South Carolina Industrial Commission requested that our committee recommend to them four consultants for their use in the evaluation of industrial injury and illness cases. This committee recommended that Dr. G. Lynn Derrick in Ophthalmology, Dr. George McLean in Plastic Surgery, Dr. Ben Miller in Internal Medicine, Dr. William Barr in Family Practice serve in this capacity as consultants to the Industrial Commission.

A meeting of our committee was held at Myrtle Beach, October 1, 1978, in which most of our members participated while attending the Annual Educational Conference sponsored by the South Carolina Industrial Commission. Members of our committee had served in planning this educational seminar with the Industrial Commission at Myrtle Beach.

Our last meeting was February 5, 1979 in Columbia.

During each of our meetings, appropriate additions and changes to the procedure codes and fees of the South Carolina Industrial Commission fee schedule were recommended.

During the years 1977 and 1978, our committee worked with the Industrial Commission in sponsoring and carrying out seminars (workshops) in various cities over the state in an effort to inform and educate physicians and their employees as well as personnel departments of industries in the procedures and forms to be used in managing the paperwork of industrial illnesses and accidents. Additional workshops are being planned for this year.

Our committee was asked by the Industrial Commission to study and make recommendations in regard to a statewide rehabilitation counseling program to assist in getting prompt and appropriate medical care to the severely injured worker and to help establish appropriate rehabilitation with the view toward minimizing time lost and severity of disability.

At the request of the Industrial Commission, our committee submitted a list of physicians specializing in pulmonary diseases throughout the state for their use as consultants.

Our committee met jointly with the South Carolina Industrial Commission in October 1978 and in February of 1979 in order to maintain a continuing dialogue related to problems encountered by both Commissioners and Industrial Commission staff and the physicians in the state in providing service to the injured worker, to industry, and to each other.

This committee offered counseling to the Medical Directors of the Industrial Commission and to the Industrial Commission itself in regard to the question of possible inappropriate medical services and inappropriate charges for services.

Though there has been some delay the Commission has informed us that the new loose leaf fee schedule will be published some time in March of 1979.

Respectfully submitted,
W. J. GOUDELOCK, *Chairman*

PERINATAL AND MATERNAL HEALTH COMMITTEE

The South Carolina Medical Association's Perinatal and Maternal Health Committee has held 4 meetings this year. All 1977 death certificates of females aged 15-44 years were screened to get an accurate count of the deaths associated with pregnancy. Approximately 800 deaths occurred in 1977 of women 15-44 years of age. Ten women who died within 42 days of pregnancy were reported, but the Committee found a total of 20 deaths indicating that there has been 100% under-reporting of maternal deaths in South Carolina. Of that 20, 16 maternal deaths were investigated by the Committee. 2 died from ectopic pregnancy, 3 died from hemorrhage, 3 died from infections, 1 died from pulmonary embolism, 2 died due to trauma, and 1 died from uncontrolled diabetes.

The 1978 death certificates of females aged 12 to 45 are currently being analyzed by the Committee. According to reported information, maternal deaths have occurred. The Committee will further investigate the information to determine if any patterns exist.

All physicians are urged to report maternal deaths to the Department of Health and Environmental Control within 24 hours. In an effort to encourage reporting by hospitals, the Committee has contacted the South Carolina Hospital Association concerning the encouragement of hospital reporting.

The Committee is also studying neonatal and fetal deaths due to asphyxia syndrome and/or meconium aspiration. Information concerning the location of the death and the quality of care the mother received will be obtained for this study.

Respectfully Submitted,
HENRY C. HEINS, JR., M.D., *Chairman*

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Brief Summary

INDICATION: Tenuate and Tenuate Dospan are indicated in the management of exogenous obesity as a short-term adjunct (a few weeks) in a regimen of weight reduction based on caloric restriction. The limited usefulness of agents of this class should be measured against possible risk factors inherent in their use such as those described below.

CONTRAINDICATIONS: Advanced arteriosclerosis, hyperthyroidism, known hypersensitivity, or idiosyncrasy to the sympathomimetic amines, glaucoma. Agitated states. Patients with a history of drug abuse. During or within 14 days following the administration of monoamine oxidase inhibitors, (hypertensive crises may result).

WARNINGS: If tolerance develops, the recommended dose should not be exceeded in an attempt to increase the effect; rather, the drug should be discontinued. Tenuate may impair the ability of the patient to engage in potentially hazardous activities such as operating machinery or driving a motor vehicle; the patient should therefore be cautioned accordingly. *Drug Dependence*: Tenuate has some chemical and pharmacologic similarities to the amphetamines and other related stimulant drugs that have been extensively abused. There have been reports of subjects becoming psychologically dependent on diethylpropion. The possibility of abuse should be kept in mind when evaluating the desirability of including a drug as part of a weight reduction program. Abuse of amphetamines and related drugs may be associated with varying degrees of psychologic dependence and social dysfunction which, in the case of certain drugs, may be severe. There are reports of patients who have increased the dosage to many times that recommended. Abrupt cessation following prolonged high dosage administration results in extreme fatigue and mental depression; changes are also noted on the sleep EEG. Manifestations of chronic intoxication with anorectic drugs include severe dermatoses, marked insomnia, irritability, hyperactivity, and personality changes. The most severe manifestation of chronic intoxications is psychosis, often clinically indistinguishable from schizophrenia. *Use in Pregnancy*: Although rat and human reproductive studies have not indicated adverse effects, the use of Tenuate by women who are pregnant or may become pregnant requires that the potential benefits be weighed against the potential risks. *Use in Children*: Tenuate is not recommended for use in children under 12 years of age.

PRECAUTIONS: Caution is to be exercised in prescribing Tenuate for patients with hypertension or with symptomatic cardiovascular disease, including arrhythmias. Tenuate should not be administered to patients with severe hypertension. Insulin requirements in diabetes mellitus may be altered in association with the use of Tenuate and the concomitant dietary regimen. Tenuate may decrease the hypotensive effect of guanethidine. The least amount feasible should be prescribed or dispensed at one time in order to minimize the possibility of overdosage. Reports suggest that Tenuate may increase convulsions in some epileptics. Therefore, epileptics receiving Tenuate should be carefully monitored. Titration of dose or discontinuance of Tenuate may be necessary.

ADVERSE REACTIONS: *Cardiovascular*: Palpitation, tachycardia, elevation of blood pressure, precordial pain, arrhythmia. One published report described T-wave changes in the ECG of a healthy young male after ingestion of diethylpropion hydrochloride. *Central Nervous System*: Overstimulation, nervousness, restlessness, dizziness, jitteriness, insomnia, anxiety, euphoria, depression, dysphoria, tremor, dyskinesia, mydriasis, drowsiness, malaise, headache; rarely psychotic episodes at recommended doses. In a few epileptics an increase in convulsive episodes has been reported. *Gastrointestinal*: Dryness of the mouth, unpleasant taste, nausea, vomiting, abdominal discomfort, diarrhea, constipation, other gastrointestinal disturbances. *Allergic*: Urticaria, rash, ecchymosis, erythema. *Endocrine*: Impotence, changes in libido, gynecomastia, menstrual upset. *Hematopoietic System*: Bone marrow depression, agranulocytosis, leukopenia. *Miscellaneous*: A variety of miscellaneous adverse reactions has been reported by physicians. These include complaints such as dyspnea, hair loss, muscle pain, dysuria, increased sweating, and polyuria.

DOSAGE AND ADMINISTRATION: Tenuate (diethylpropion hydrochloride): One 25 mg. tablet three times daily, one hour before meals, and in midevening if desired to overcome night hunger. Tenuate Dospan (diethylpropion hydrochloride) controlled-release: One 75 mg. tablet daily, swallowed whole, in midmorning. Tenuate is not recommended for use in children under 12 years of age.

OVERDOSAGE: Manifestations of acute overdosage include restlessness, tremor, hyperreflexia, rapid respiration, confusion, assaultiveness, hallucinations, panic states. Fatigue and depression usually follow the central stimulation. Cardiovascular effects include arrhythmias, hypertension or hypotension and circulatory collapse. Gastrointestinal symptoms include nausea, vomiting, diarrhea, and abdominal cramps. Overdose of pharmacologically similar compounds has resulted in fatal poisoning, usually terminating in convulsions and coma. Management of acute Tenuate intoxication is largely symptomatic and includes lavage and sedation with a barbiturate. Experience with hemodialysis or peritoneal dialysis is inadequate to permit recommendation in this regard. Intravenous phentolamine (Regitine®) has been suggested on pharmacologic grounds for possible acute, severe hypertension, if this complicates Tenuate overdosage.

Product Information as of April, 1976

MERRELL-NATIONAL LABORATORIES Inc.
Cayey, Puerto Rico 00633
Direct Medical Inquiries to:
MERRELL-NATIONAL LABORATORIES
Division of Richardson-Merrell Inc.
Cincinnati, Ohio 45215, U.S.A.

Licensor of Merrell®

References: 1. Citations available on request—Medical Research Department, MERRELL RESEARCH CENTER, MERRELL-NATIONAL LABORATORIES, Cincinnati, Ohio 45215. **2**. Hoekenga, M.T., O'Dillon, R.H., and Leyland, H.M.: A Comprehensive Review of Diethylpropion Hydrochloride. International Symposium on Central Mechanisms of Anorectic Drugs, Florence, Italy, Jan. 20-21, 1977.

Merrell

8-3921 (Y587A)

RESOLUTIONS TO BE PRESENTED AND VOTED ON AT THE ANNUAL MEETING

SUBMITTED BY: CHARLESTON MEDICAL SOCIETY
SUBJECT: CARE OF PROFOUNDLY HANDICAPPED CHILDREN

WHEREAS, we believe that the care of the profoundly handicapped children of this State should be provided for; and

WHEREAS, the present arrangement of school teachers being made responsible for the health care of profoundly retarded children, many with very severe health problems, is inappropriate and actually dangerous to the child; and

WHEREAS, the Department of Mental Retardation is by the nature of their many programs a much more appropriate agency to deal with these profoundly handicapped children;

BE IT RESOLVED that all efforts be made to encourage the Department of Mental Retardation to assume the responsibility for setting up programs for these profoundly mentally retarded children, with the Department of Education acting in an advisory role as to any possible educational or training programs which may benefit these children.

SUBMITTED BY: SUMTER-CLARENDON MEDICAL SOCIETY
SUBJECT: BILLING FOR AND PAYMENT BY STATE AGENCIES

WHEREAS several state agencies require bills sent to them in a matter of days from the date of charge and usually at the end of their fiscal year and

WHEREAS: in some cases it is not known that the patient is even covered by such programs and/or it is sometimes difficult in getting signatures or proper identification numbers and

WHEREAS: this extremely short period required to have bills sent to these agencies brings an undue hardship to the physicians and their offices and

WHEREAS: this seems to be an internal accounting and budget problem for the state agencies and

WHEREAS: we physicians rarely, if ever, receive payment in such a time frame as they require the billing, be it therefore

RESOLVED: that the South Carolina Medical Association through its committees to the state agencies and also through council, notify these agencies that a longer time is needed in some cases before their deadline for such billings is reached, and payment should be made within a same reasonable time period whenever a bill is sent for procedures performed.

SUBMITTED BY: WILLIAMSBURG COUNTY MEDICAL SOCIETY
SUBJECT: UTILIZATION REVIEW

WHEREAS, the members of the South Carolina Medical Association who are members of medical staffs of hospitals in South Carolina fully agree and support the concept of *peer review*,

BE IT RESOLVED, that such members will continue such *peer reviews* as outlined by the South Carolina Professional Standards Review Organization (PSRO); and

WHEREAS, the members of the South Carolina Medical Association who are members of medical staffs of hospitals in South Carolina do *not* believe that *hospital utilization review* is a legitimate function of the *medical staff* of any hospital,

BE IT RESOLVED, 1. That the South Carolina Medical Association recommends that the present physician advisors for hospital *utilization review* relinquish their positions immediately, and that other staff members knowingly refrain from performing this function. The hospital *utilization review* should be performed by the *insurance providers*.

2. That the termination letter or other notification to the patient, his family, or responsible party should not contain any reference to the attending physician, local medical staff, or other local physician review organization. It is our opinion that any termination notification should be carried out by the *insurance carrier* or *intermediary*.

After specializing in the treatment of alcoholism and drug addiction for 17 years, we found . . .

if there are problems and there is drinking... drinking may be the only problem!

Willingway Hospital

BOX 508 STATESBORO, GA 30458 (912) 764-6236

Accredited by the Joint Commission on Accreditation of Hospitals

ANNUAL MEETING SPEAKER

CHARLES MAX COLE, M.D.
Member, Board of Trustees, AMA

Charles Max Cole, M.D., will address the SCMA House of Delegates on Friday, April 27 at 9:30 a.m. in the Center and West Ballrooms of the Myrtle Beach Hilton. He will speak to Council on April 26 at 4:00 p.m., and the membership is invited to attend.

A general surgeon in Dallas, Texas, Dr. Gardner is a member of the Board of Trustees of the American Medical Association.

In addition to his present position with the AMA, Dr. Cole has been a Delegate from the Texas Medical Association to the AMA and has been a representative from the AMA to the National Joint Practice Commission. He is a Past President and a Past Speaker of the House of Delegates of the Texas Medical Association. His offices with the Dallas County Medical Society have included those of Secretary, Vice President and President.

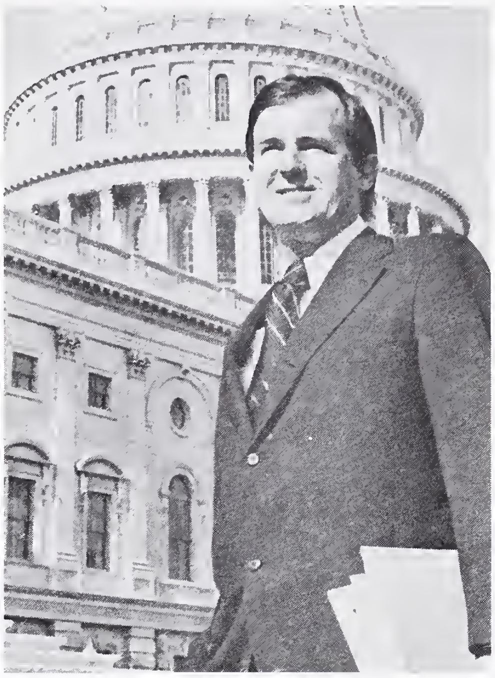
Born in Garland, Texas, Dr. Cole completed his Undergraduate education at Southern Methodist University, with a B.A. Degree in 1937. He attended the University of Iowa Medical School, receiving his M.D. Degree in 1942. He took internship and residency training at Parkland Memorial Hospital in Dallas in 1942-44, and in 1948.

Dr. Cole is a Diplomate on the American Board of Surgery. Since 1970, he has had an academic appointment as Clinical Professor of Surgery at the University of Texas Southwestern Medical School. Hospital appointments include Chief of Surgery at Presbyterian Hospital of Dallas; Former Chief of Surgery, Medical Arts Hospital of Dallas; St. Paul Hospital; Baylor Hospital; and President of the Attending Staff at Parkland Hospital, 1967-68. Dr. Cole was a Captain in the Medical Corps, United States Army, 1945-47.

Active in community affairs for many years, Dr. Cole has served on numerous Boards and held office in various civic organizations. Special awards he has received include the Dallas Hospital Council Health Award and the North Dallas Chamber of Commerce Award for Excellence in Medicine. He is a member of the University Park Methodist Church and the Rotary Club of Dallas.

Dr. Cole and his wife, Marilynn, have two daughters, Cynthia and Karen.

SOCPAC LUNCHEON SPEAKER



CONGRESSMAN BUTLER DERRICK

Congressman Butler Derrick is the 1979 Speaker at the SOCPAC Luncheon to be held at 1:15 p.m., Saturday, April 28, 1979 in the West Ballroom of the Myrtle Beach Hilton.

Congressman Derrick, of Edgefield County, South Carolina, is serving his third term as representative for the Third Congressional District of South Carolina. He was first sworn in the Congress on January 14, 1975, and was re-elected in 1976 and 1978.

He was born September 30, 1936, and is the son of Mary English (Scott) and the late Butler Carson Derrick, Sr., of Johnston, S. C. Congressman Derrick attended the University of South Carolina and was graduated from the University of Georgia Law School in 1965.

Congressman Derrick is married to the former Suzanne Mims of Edgefield, and they have two children, Lydia Gile and Butler Carson, III. They are members of the Trinity Episcopal Church in Edgefield.

Prior to his election to the 94th Congress, Congressman Derrick practiced law in Edgefield and served six years as a member of the South Carolina House of Representatives. He was recognized by the South Carolina Jaycees Association as the Outstanding Man of the Year in 1971-72.

In Washington, he served two terms on the House Budget Committee, being the first freshman in the Congress ever to receive such an assignment. As Past Chairman of the Task Force on the Budget Process, he headed a panel that had oversight over all Federal budgetary matters. He has also served on the House Committee on Banking, Finance and Urban Affairs, and on two of its sub-committees. He now serves on the House Rules Committee.

Currently, he is treasurer of the 94th Caucus, and serves as the President of the Edgefield County Fish and Game Association. A nationally recognized advocate of conservation of natural resources, he was named by the S. C. Wildlife Federation as the "South Carolina Conservationist of the Year" for 1977. The National Wildlife Federation selected him as the "National Conservationist of the Year" for his actions in 1977 in natural resource areas.

He was awarded an honorary Doctor of Humanities degree for "achievement as a citizen and public servant" by Lander College, Greenwood, S. C., in February, 1978.

...in the functional bowel/irritable bowel syndrome*

Bentyl®

(dicyclomine hydrochloride USP)

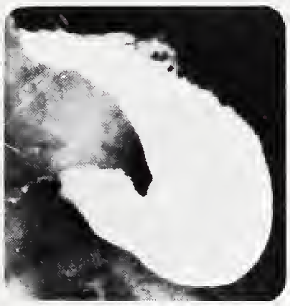
10 mg. capsules, 20 mg. tablets,
10 mg./5 ml. syrup, 10 mg./ml. injection

helps control abnormal motor activity with minimal anticholinergic side effects†

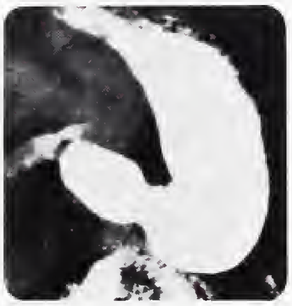
Demonstrated smooth muscle relaxant activity.

In this double-blind study, twenty patients having G.I. series and exhibiting spasm were randomly selected to receive either 2 cc. of Bentyl or sodium chloride intramuscularly. Ten minutes after the injection another radiograph was taken . . .

. . . Bentyl produced definite relaxation in 8 of 10 patients. The sodium chloride produced relaxation in only 3 of 10. No side effects occurred in either group of patients.



Pylorospasm has almost totally blocked passage of barium meal.



Barium meal beginning to pass 10 minutes after intramuscular injection of 20 mg. Bentyl.

"The correlation of spasm relief and drug given was excellent."

*This drug has been classified "probably" effective in treating functional bowel/irritable bowel syndrome.

†See Warnings, Precautions and Adverse Reactions.

See following page for prescribing information.

Reference:
King, J.C. and Starkman, N.M.: Evaluation of an antispasmodic. Double-blind evaluation to control gastrointestinal spasms occurring during radiographic examination. A preliminary report. Western Med. 5:356-358, 1964.

Merrell

8-4420 (Y736A) MNR-804

Bentyl®
(dicyclomine hydrochloride USP)
Capsules, Tablets, Syrup, Injection
AVAILABLE ONLY ON PRESCRIPTION
Brief Summary

INDICATIONS
Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the following indications as "probably" effective:
For the treatment of functional bowel/irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis.
THESE FUNCTIONAL DISORDERS ARE OFTEN RELIEVED BY VARYING COMBINATIONS OF SEDATIVE, REASSURANCE, PHYSICIAN INTEREST, AMELIORATION OF ENVIRONMENTAL FACTORS.
For use in the treatment of infant colic (syrup).
Final classification of the less-than-effective indications requires further investigation.

CONTRAINDICATIONS: Obstructive uropathy (for example, bladder neck obstruction due to prostatic hypertrophy); obstructive disease of the gastrointestinal tract (as in achalasia, pyloroduodenal stenosis); paralytic ileus, intestinal atony of the elderly or debilitated patient; unstable cardiovascular status in acute hemorrhage; severe ulcerative colitis; toxic megacolon complicating ulcerative colitis; myasthenia gravis. WARNINGS: In the presence of a high environmental temperature, heat prostration can occur with drug use (fever and heat stroke due to decreased sweating). Diarrhea may be an early symptom of incomplete intestinal obstruction, especially in patients with ileostomy or colostomy. In this instance treatment with this drug would be inappropriate and possibly harmful. Bentyl may produce drowsiness or blurred vision. In this event, the patient should be warned not to engage in activities requiring mental alertness such as operating a motor vehicle or other machinery or perform hazardous work while taking this drug. PRECAUTIONS: Although studies have failed to demonstrate adverse effects of dicyclomine hydrochloride in glaucoma or in patients with prostatic hypertrophy, it should be prescribed with caution in patients known to have or suspected of having glaucoma or prostatic hypertrophy. Use with caution in patients with: Autonomic neuropathy. Hepatic or renal disease. Ulcerative colitis. Large doses may suppress intestinal motility to the point of producing a paralytic ileus and the use of this drug may precipitate or aggravate the serious complication of toxic megacolon. Hyperthyroidism, coronary heart disease, congestive heart failure, cardiac arrhythmias, and hypertension. Hiatal hernia associated with reflux esophagitis since anticholinergic drugs may aggravate this condition.
Do not rely on the use of the drug in the presence of complication of biliary tract disease. Investigate any tachycardia before giving anticholinergic (atropine-like) drugs since they may increase the heart rate. With overdosage, a curare-like action may occur. ADVERSE REACTIONS: Anticholinergics/antispasmodics produce certain effects which may be physiologic or toxic depending upon the individual patient's response. The physician must delineate these. Adverse reactions may include xerostomia; urinary hesitancy and retention; blurred vision and tachycardia; palpitations; mydriasis; cycloplegia; increased ocular tension; loss of taste; headache; nervousness; drowsiness; weakness; dizziness; insomnia; nausea; vomiting; impotence; suppression of lactation; constipation; bloated feeling; severe allergic reaction or drug idiosyncrasies including anaphylaxis; urticaria and other dermal manifestations; some degree of mental confusion and/or excitement, especially in elderly persons; and decreased sweating. With the injectable form there may be a temporary sensation of lightheadedness and occasionally local irritation. DOSAGE AND ADMINISTRATION: Dosage must be adjusted to individual patient's needs.
Usual Dosage: Bentyl 10 mg. capsule and syrup: *Adults:* 1 or 2 capsules or teaspoonfuls syrup three or four times daily. *Children:* 1 capsule or teaspoonful syrup three or four times daily. *Infants:* ½ teaspoonful syrup three or four times daily. (May be diluted with equal volume of water.) Bentyl 20 mg.: *Adults:* 1 tablet three or four times daily. Bentyl Injection: *Adults:* 2 ml. (20 mg.) every four to six hours intramuscularly only. NOT FOR INTRAVENOUS USE. MANAGEMENT OF OVERDOSE: The signs and symptoms of overdose are headache, nausea, vomiting, blurred vision, dilated pupils, hot, dry skin, dizziness, dryness of the mouth, difficulty in swallowing, CNS stimulation. Treatment should consist of gastric lavage, emetics, and activated charcoal. Barbiturates may be used either orally or intramuscularly for sedation but they should not be used if Bentyl with Phenobarbital has been ingested. If indicated, parenteral cholinergic agents such as Urecholine® (bethanecol chloride USP) should be used.
Product Information as of October, 1978.

Injectable dosage forms manufactured by CONNAUGHT LABORATORIES, INC., Swiftwater, Pennsylvania 18370 or TAYLOR PHARMACAL COMPANY, Decatur, Illinois 62525 for MERRELL-NATIONAL LABORATORIES, Division of Richardson-Merrell Inc., Cincinnati, Ohio 45215, U.S.A.

Merrell
MERRELL-NATIONAL LABORATORIES
Division of Richardson-Merrell Inc
Cincinnati, Ohio 45215 U.S.A.

REPORT OF THE EDITOR OF *THE JOURNAL*

The future of scientific journals in this country has been threatened by the announced intent of the Internal Revenue Service to revoke the tax-exempt status of most of our periodicals. Recent policy trends of the United States Post Office and of the Interstate Commerce Commission further jeopardize the viability of journals. The editor of *The Journal of the American Medical Association* summarized our situation: "If government persists in its attacks on scientific journals, it will find not the golden eggs it seeks, but as described in Aesop's fable: 'Thinking to get at once all the gold the goose could give, he killed it and opened it only to find — nothing.' "

Our Journal celebrates its 75th volume. We look back upon a heritage of continuous publication of material which we think has served the people of our state. Nevertheless, rising printing costs and changing trends of medical authorship combine with the new thrusts of government agencies to threaten the viability of state journals such as ours. We share the sentiment of the first editors of our journal, who stated in their opening editorial: "The editors fully realize the arduous nature of the undertaking, and urge upon every man the importance of contributing his share." Also, like the original editors, we "will welcome the frankest criticism." This remains *your* journal; we welcome *your* thoughts and contributions.

Respectfully submitted,
Charles S. Bryan, M.D.

Editorials

SUNSET ON THE AGE OF KEFLIN

The tasteful two-page advertisement makes use of one of man's oldest and most powerful metaphors: the evening of the day. The setting of a brilliant sun over a calm sea occupies an entire page, reminding us in relatively small letters, beneath the horizon, of "Keflin — cephalothin sodium." On the facing page, the first rays of dawn emerge over a gently undulating countryside, telling us, at the point of their convergence, of a new day: "Mandol I.M./I.V. — cefamandole naftate." Across this friendly sky stands the message: "a new horizon in cephalosporin therapy."

The new horizon represents what is being called the "second generation" of cephalosporin antibiotics. The "first generation" began with cephalothin (Keflin), the useful outcome of the discovery of *Cephalosporium acremonium* near a sewage outlet in 1945. Numerous similar drugs cropped up in a decade of keen competition. Several of these — most notably cephapirin (Cefadyl, Bristol) and cephradine (Velosef, Squibb), proved to be virtually identical to cephalothin but offered no major advantages. During this decade, however, one fact became clear: the cephalosporins emerged as the single major item in most hospital pharmacy budgets. At a representative hospital, cephalosporins might comprise 25 percent of the total expenditure for drugs in a given period.

The new horizon of "second generation" cephalosporins now consists of cefamandole (Mandol, Lilly), and cefoxitin (Mefoxin, Merck, Sharp, and Dome); the latter drug is actually a *cephamycin*, being derived from a *Streptomyces* rather than a *Cephalosporium*, but for practical purposes can be regarded as another cephalosporin. Other agents, notably cefuroxime and HR 756, are in developmental phases. These new agents offer a single advantage: they inhibit growth of certain gram-negative bacteria which are resistant to cephalothin and the other "first generation" agents. Since most cephalosporin usage is either "preventive" or "broad-spectrum, empiric," it follows that these newer agents should by and large replace the older ones for nearly all situations.

But what about *cost*?

President Carter, in his State of the Union address, singled out control of hospital costs as the single major factor for preservation of our economy. Although many hospital cost items are beyond the control of practicing physicians, some *are* within our control — most notably, utilization of diagnostic tests and procedures and utilization of drugs.

At the time of this writing, the quoted cost of cephalothin (Keflin, Lilly) to our hospital pharmacy is $2.41 per gram. The quoted cost of cefamandole (Mandol, Lilly) is $4.81 per gram.

Cephamandole was released in the fall of 1978. Usage in our community hospital increased as follows:

	Grams Used	Dollar Amount
October, 1978 . .	0	0
November, 1978	409 grams	$1790.78
December, 1978	1456 grams	$6376.52
January, 1979 . .	2150 grams	$9411.50

By January 1979, cefamandole surpassed cephalothin in grams used, and the total expenditure for cefamandole more than doubled that for cephalothin. Overall expenditure for cephalosporins increased 82 percent above that for the previous year, and almost all of the excess could be attributed to use of the new drug, cefamandole (Mandol). Findings at another hospital in the same community were virtually identical.

Recently, the South Carolina Medical Association's House of Delegates passed a resolution on cost control, submitted by Dr. Edmund R. Taylor of the Columbia Medical Society, which seems pertinent to this issue. Noting the problems of voluntary cost control, it was resolved that various groups concerned with health care should be "sitting down in each hospital, in good faith, to sink their collective teeth into this problem by opening up close communication and cooperation with the specific aim of reducing costs, when not detrimental to sound patient care. . . ." It follows that, when a new but more expensive drug is introduced as a "replacement" for an older agent, medical staffs should discuss the issue and decide whether the cost-benefit ratio is favorable.

Remarkably, the experience with cefamandole cited above came without approval of the agent by the hospital Pharmacy and Therapeutics Committee, and without routine reporting of cefamandole susceptibilities by the hospital bacteriology laboratory. Surveys indicated that some gram-negative rods isolated were, in fact, resistant to cephalothin and susceptible to cefamandole (or cefoxitin), but in very few instances was choice of cefamandole based on such findings. Indeed, ongoing studies suggest that cephalothin-resistant, cefamandole-susceptible pathogens appear in a few, well-defined situations (such as patients who have been in the hospital for a week or longer, patients who have been receiving antibiotics, or patients with prior urinary tract infections and instrumentation). In short, it seems likely that cephalothin (or cefazolin) could have been employed in most situations with equally favorable results.

These issues are often obfuscated by the numerous subtle details about drugs which are advanced in favor of one or another agent. For instance, it is stated that the dosage of cefamandole, compared to cephalothin, can be lower, and therefore cost might be equivalent even though cefamandole is more expensive per gram. However, the dosage indicated for "seriously ill" patients is the same for both agents. In the experience cited above cefamandole usage *did* add to total hospital costs. Another argument for cefamandole claims that the agent has "good anaerobic activity." However, the concentration of cefamandole needed to inhibit growth of *Bacteroides fragilis* is quite high — in one study only 57 percent of strains were inhibited by a concentration of 32 mcg/ml — and therefore it seems extremely doubtful that cefamandole could be chosen for *reliable* therapy of anaerobic infections below the diaphragm.[1]

It seems difficult to argue with the recent conclusion of *The Medical Letter*[2]:

> "For treatment of gram-negative bacillary infections, both of these agents (cefamandole and cefoxitin) have a broader spectrum than previously available cephalosporins, but aerobic gram-negative enteric pathogens are more likely to be susceptible to gentamicin or tobramycin, and anaerobes to chloramphenicol or clindamycin. Until more data become available, both cefamandole and cefoxitin should generally be reserved for special situations in which they may be preferred because of their relative safety or because of bacterial resistance to better established drugs."

The cephalosporins will remain useful for preventive ("prophylactic") antibiotic therapy in surgery, but for most patients *cefazolin* (Kefzol; Ancef) — with its lower cost and extremely favorable pharmacokinetics — would seem preferable to either of the two new drugs.

We must resist the temptation to blame the pharmaceutical industry for promoting its products. Development of new drugs is extremely expensive, and it can be argued that a limit on drug costs is ultimately placed by the competition in the open market. Rather, physicians at local levels must become more involved in these issues, in keeping with the recent SCMA resolution. The alternatives — most notably compulsory health insurance and a new host of regulations — were spelled out clearly in the resolution. The sun setting on Keflin, in the advertisement, could also symbolize the waning of the age of free enterprise in health care delivery as we know it.

— CSB

REFERENCES

1. Ernst EC, Berger S, Barza M, Jacobus NV, Tally FP: Activity of cefamandole and other cephalosporins against aerobic and anaerobic bacteria. *Antimicrob Agents Chemother* 9: 852-855, 1976.
2. Cefamandole and Cefoxitin. *The Medical Letter* vol. 21, no. 3 (February 9, 1979)

LETTER TO THE EDITOR

To The Editor:

Your editorial on beepers was timely and pertinent and was certainly appreciated by me. My only complaint is that it did not damn these instruments sufficiently. A facet you failed to mention is that users of beepers seem to be forgetting common courtesy when they permit their beeper, for whatever reason, to interrupt a speaker or someone else who at a meeting has the right to the floor.

I can conceive of no situation where it is necessary that beepers go off duirng a meeting for surely a simple phone call alerting the answering service as to the location of the meeting would make it possible for them to hold calls or send someone in person to deliver a message.

Thanks again for the article. Lets trust that a "thoughtful decision" as well as courtesy may return to the "instantly available physician."

Sincerely,
Lawrence V. Jowers, M.D.
1634 Taylor Street
Columbia, S. C. 29201

A symposium on various aspects of hypertension will be presented April 19th in the Richland Memorial Hospital Auditorium from 12:00 noon to 5:00 p.m.

"Hypertension Update - 1979" is sponsored by the Columbia Medical Society, the University of South Carolina School of Medicine and the Family Practice Residency at Richland Memorial Hospital. It will be funded by means of an educational grant from Pfizer Laboratories.

This should be an excellent local program and an effective method of learning (and obtaining CME credits) for physicians in South Carolina who are interested in hypertension.

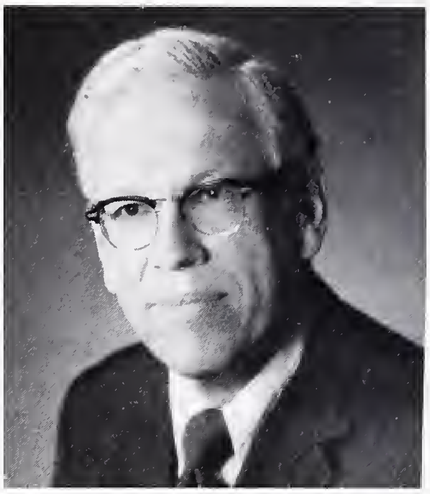
SOUTH CAROLINA CHAPTER OF AMERICAN COLLEGE OF EMERGENCY ROOM PHYSICIANS
SATURDAY, APRIL 28, 1979
MYRTLE BEACH HILTON
MYRTLE BEACH, S. C.

11:00 a.m.-1:00 p.m.	Board of Directors Meeting and Luncheon — Parlour IV
1:00 p.m.	SCIENTIFIC SESSION — Center Ballroom Coordinator: Warren B. Garrison, M.D., Director of Education for Emergency Medicine Residency
1:00-1:45 p.m.	*Burns to the Esophagus* — Foster Marshall, M.D.
2:00-2:45 p.m.	*Evaluation of Potential Suicide* — John M. Foxworth, M.D.
3:00-3:30 p.m.	*Assessment of Trauma — Special Radiological Procedures* — Ivan Vujic, M.D.
4:00-4:45 p.m.	*Treatment of the Dialysis Patient in the Emergency Room* — James I. Raymond, M.D.

JOIN THE SCMA TODAY

Send dues statement and check to:
P. O. Box 11188
Columbia, S. C. 29211

President's Pages



IT SEEMS ONLY YESTERDAY —

It's hard to take in that the year is almost over. It seems only yesterday — really, it does — that I was trying to compose my first President's Page for the May, 1978 issue of *The Journal*. That's when I learned about such things as deadlines for the next issue and length restrictions, but also began to appreciate the many talents of Joy Drennen, our Managing Editor. Putting *The Journal* together is just one *one* of her duties. She accomplishes them all with amazing competence and with the sort of finesse which enables her to needle you almost painlessly into getting your part of the job done on time. Many thinks, Joy!

A year has passed, and I am told (by Joy) that I am composing my last President's Page. Even though it does seem only yesterday, I must confess that writing this page sometimes is a real chore. At times, words just do not flow at the speed one desires, and it takes thought and concentration to produce even a short article which may hopefully be worth reading. I trust I have managed to issue a few worthwhile challenges, to stimulate some thought on critical issues and to direct the Association into proper attitudes and activities.

This last President's Page should be, I suppose, a place for reflection upon what has occurred and has been accomplished during the past year, and then, more importantly, a glimpse of the future.

It has been an eventful year. While there has been a continuing assault on the "health care industry," by many in government and in the news media, we in the medical profession in this country have still managed to survive with more individual freedoms to practice as we feel competent and in the manner we think best for our patients, than most anywhere in the world.

I will mention only briefly a few of the things of importance to physicians which have happened during the past twelve months:

1. *NHI:* The refusal by the AMA House of Delegates in December to endorse an NHI Bill to me was the most important development in organized medicine of the past year.

2. *Cost Containment:* The success of the Voluntary Effort of the AMA, AHA, and FAH has been heartening, and must be publicized; the further pursual of the VE must be used as a continuing argument against the restrictive, unfair, mandatory levels of cost containment sought by President Carter and others in government.

3. *SCMA Delegation Meeting with Congressional Leaders in Washington, January 23, 1979:* This was a "first" as far as our relations with our Congressional Delegation are concerned, and I believe represented a major forward step in presenting our views to our representatives in Washington. It was preceded by an SCMA presentation to the Congressional Delegation at its annual public forum in Columbia on January 9. Considerable favorable feedback has already been received. Both should be repeated each year.

4. *SCMA Relationships with Other Organizations:* Our liaison with other organizations in the health care field, including the SCHA, SCNA, the S. C. Pharmaceutical Association, DHEC, DSS and the

Board of Medical Examiners has been increased during the past year and is of critical importance in the future. In addition, a liaison committee with the South Carolina Bar has been re-activated.

5. *Second Opinion Surgery Programs:* Such programs on a mandatory basis have been continually opposed by physicians in South Carolina, and by a majority of the medical profession over the country. Hopefully, these programs will continue to falter and will soon fall by the wayside.

6. *HMOs:* Despite subsidization by federal funds, there has not been the rapid expansion of HMOs predicted when the Health Maintenance Act was passed in late 1973. Even with additional protective and supportive legislation, growth of these organizations either in the form of group practice associations or individual practice associations (IPAs), has been slow. By August, 1978, there were only approximately 200 HMOs established, instead of the 1700 predicted for two years earlier. However, the impetus for either HMOs or IPAs may become more pervasive in South Carolina during the coming years and, therefore, all physicians would be well advised to read the series of articles on this subject in the March, 1979 issue of *Private Practice.*

7. *HSAs:* The effect of HSA decisions on the delivery of health care in South Carolina has become more apparent during the past year. Physicians, both individually and through county society committees, are monitoring HSAs more carefully and, hopefully, will be able to modify and direct HSA decisions appropriately.

These are just a few of the highlights. You may think of many more. What does the future hold?

Harrison Peeples will become the new President on April 28 at the SCMA Presidential Inauguration and Special Awards Presentations Ceremony at Myrtle Beach. This occasion will be in a new format on which Harrison and I have worked together. We hope it will be favorably received by the membership and will be a memorable event. Certainly, in Harrison Peeples, the SCMA will have an outstanding leader for the coming year. His past accomplishments are many. As President-Elect, during the past year, he chaired the Membership Committee which successfully brought our membership level above 2,000 for the first time and which has established membership policies and recruitment programs which will undoubtedly take us to greater heights during the coming year.

His task will not always be easy, however. Many problems for our state Association remain only partially solved and present challenges for the future: Continuing Medical Education, the Impaired Physicians' Program, the Medical Liability Insurance Program, Chiropractic Bills, and legislation to expand Optometry are among the problems which will need to be addressed further. Under Harrison Peeples' leadership, with the support of a hardworking Council and Executive Committee, and with the active participation of the membership in important committee assignments, the SCMA will, I feel sure, achieve monumental progress during the coming year.

I cannot closed without expressing a few words of special thanks:

. . . to the entire SCMA staff who have given me every possible assistance and cooperation;

. . . to all the members of Council, whose dedication to the Association and to the people of South Carolina is unceasing;

. . . to the committee Chairmen and committee members who accepted the responsibilities accorded them and who, during the past year, have made our committee system increasingly effective;

. . . to the Auxiliary, for their unfailing support and for their imaginative and rewarding projects; and, finally,

. . . to the House of Delegates for its attention to the affairs of the Association and for affording me the privilege of serving as your President. It has been a most rewarding experience, and an honor for which I am indeed grateful.

John C. Hawk, Jr., M.D.,
President

1978 INTERIM MEETING OF THE AMA HOUSE OF DELEGATES

DECEMBER 6, 1978
CHICAGO, ILLINOIS
Harrison L. Peeples, M.D., Delegate

The Interim meeting of the AMA House of Delegates was held in the cold windy city of Chicago, December 6, 1978. The Clinical-Scientific sessions are held on regional basis with the current one being held in Las Vegas.

Attending the meeting were your delegates and alternates: Tucker Weston and wife, Polly; Waitus Tanner and wife, Jennie; Dessie Gilland; my wife, Lib, and I. Our president, Dr. John Hawk, was present and participated with us at the reference committee. John and others' efforts to encourage involvement by House Staff (residents) in organized medicine activities seem to be reaching fruition. We were fortunate to have the following House Staff with us: Dr. Stewart Haskin, Orthopedics, MUSC; Dr. Eric Jones, Psychiatry, MUSC; Dr. Vann Beth Schuler, Family Practice, Spartanburg; Dr. Michael Hoffman, OB-GYN, Greenville; Dr. Rick Richards, F.P., Spartanburg.

I am impressed by the interest shown by this group of young men and women and would encourage involvement of others. We need input from them and they need input from us. It is to them that the future belongs. Discussions being made now will influence the practice of medicine for the future.

About 200 items of business were considered and this particular meeting presented many difficult and far-reaching decisions for your delegates. Those actions will have a decided impact on the future course of the Association and on each individual physician.

The House currently consists of 271 delegates from 55 states and territories and more than 50 national specialty societies. The House also includes one voting delegate from the medical student, housestaff, medical schools, and the services, i.e., the Army, Air Force, Navy, V.A., and Public Health Service.

At the Interim Meeting the House opens on Sunday afternoon with reports from the AMA President, the Auxiliary, AMPAC, and AMA-ERF. Also at this time, the Dr. Benjamin Rush Award for Citizenship and Community Service is presented. This year's recipient was Doctor Tim Lee Carter, a U. S. Congressman for many years from Kentucky, who brought his medical knowledge and experience to bear on virtually every piece of major health legislation in recent years.

In order to cover the various reference committees, we divided this responsibility between us. Categoric assignments are made and reported in the following subsections by assignees as follows:

I — *Legislation; Scientific and Public Health* — Tucker Weston
II — *Insurance and Medical Service; Constitution and By-Laws* — Dessie Gilland
III — *Board of Trustee Reports; Hospital and Medical Facilities* — Waitus Tanner
IV — *Medical Education; Miscellaneous* — Harrison L. Peeples

The Council on Medical Education brought several reports before the House, and foremost of these were the number of primary care residents entering primary care practices upon completion of residency training. It notes that in 1971 only 38.7% of graduates entered primary care (I.M., P.D., OBG, F.P., G.P.) while in 1974, 56.5% entered primary care residencies. This is a significant change but does not in itself answer the matter of training in primary care. Many of the residents entering a primary care field the first year do so enroute to one of the specialties, so the significant figure is how many of these entering primary care training actually continue in primary care upon completion of their residency program. The Council on Medical Education is to make annual reports to the House in this regard.

In response to recommendation #35 and #36, from the National Commission on the Cost of Medical Care, and regarding loan forgiveness and recruitment of underserved areas, the Council pointed out that present evidence does not support emphasizing loan forgiveness, whether with punity repayment clauses or in conjunction with rural recruitment, as an effective way to correct the long range physician deficit in underserved areas. This study may be of importance to DHEC in implementation of our loan program for students in South Carolina.

Report #21, the National Commission on the Cost of Medical Care, encouraged the development of guidelines other than on a national basis to determine what medical personnel and facilities are needed in various areas to meet the expected demands of health services. The House concurs in this recommendation and has adopted the principal that "to achieve the aims of the Federation — assuring that health planning is responsive to the needs of patients in the medical profession — medical societies must become politically active and involved in all levels of the planning activity." To this aim there has been an Ad Hoc committee to develop a Federation guidebook on health planning for use by states and county medical societies. This medical services guidebook is anticipated for completion in early 1979 and should assist states and local medical societies to participate effectively in health planning activities and to protect and preserve the quality of health care in the community.

Several Resolutions addressed the issue of State Medical Societies accrediting continuing medical education programs within the state rather than on a national basis. Substitute Resolution was offered by the Reference Committee which affirms the concept of accreditation of continuing medical education by State Medical Societies whose continuing education standard meets the AMA or the Liaison Committee on Continuing Medical Education standards.

The subject of continuing education is one of great significance and is one that our association is attempting to address at this moment. This support for state involvement from the AMA comes at an appropriate time.

A report given to the House by the AMA-ERF points out that since the program began in 1962 the foundation has arranged and guaranteed almost 90 million dollars in loans for medical students and physicians in training and residency programs. There are now 400 million dollars in loans outstanding. During the first nine months of 1978, the AMA-ERF guaranteed $4.6 million in new loans. This is a worthwhile effort of the AMA and it has had outstanding support from the Auxiliary which works diligently in collecting these monies to aid students and residents.

Of these loans made by AMA-ERF, 94% have repaid them on schedule. Of the 6% who defaulted, half subsequently repaid their loan. Slow payments caused the largest number of defaults and, upon investigation, compelling reasons usually are discovered. Patience, when appropriate, and persistent follow-up result in eventual payment in most cases.

Dessie Gilland's report follows. Reports from Doctors Tanner and Weston will be published at a later date.

REPORT FROM J. D. GILLAND, M.D.

My assignment was to monitor and speak when appropriate at Reference Committees on Amendments to the Constitution and By-Laws and on Insurance and Medical Services.

The Reference Committee on Constitution and By-Laws was chaired by Dr. George L. Collins, Jr. of New York City. The majority of its time was devoted to the problems about our present Principles of Medical Ethics and its proposed revision. The controversy was centered around our relationship with the Chiropractors and our position on Chiropractic. Our Principles of Medical Ethics adopted by the House of Delegates in 1957 states in part: "A Physician should practice a method of healing founded on a scientific basis; and he should not voluntarily associate professionally with anyone who violates this principle." The Judicial Council in its *Opinions and Reports* is deeply involved in the interpretation of our Code of Ethics. Much of the controversy centers around and was precipitated by the current multiple litigations between the AMA and the Chiropractic Association. The Chiropractors allege our adherence to our Code of Ethics constitutes restraint of trade which is the basis for litigation and if successfully pursued could cost the AMA triple indemnity approaching fifty million dollars. AMA legal counsel feels confident that we will successfully defend our position. The Reference Committee recommended and the House of Delegates adopted the referral of these problems to the already constituted Ad Hoc Committee on the Principles of Medical Ethics to continue its study and to report to the House at the 1979 Annual Meeting.

The Reference Committee on Insurance and Medical Services was chaired by Dr. Patrick J. V. Corcoran of Indiana. On HMOs, the House continued to approve the concept of neutral public policy and fair market competition among all systems of Health Care Delivery. The cost of medical care was addressed by several resolutions and the House of Delegates established its position by resolving, "that the AMA recognizes that medical and health care costs have increased in part because of the increased demand for services as a result of third party payment, the number of regulations generated by federal and state agencies, professional liability insurance premiums, and because of deficit spending by the federal government."

To understand the role of AMA as the leading organized body of our profession at the national level, one has to attend and witness the proceedings of the House of Delegates to appreciate its role as it personally affects each one of us. I beseech each of you in the early years of your private practice to take the time to attend one of the AMA meetings, for I can assure you that you will be proud and supportive of the massive activities of our American Medical Association.

CONCLUSION

There were three major issues which dominated the debate and were of major interest to the House Delegates.

(1) The question of National Health Insurance and whether the AMA should cause a proposal to be introduced in the Congress, and, if so, what would be contained in the Bill.

(2) The proposed revision of the Principals of Medical Ethics and its implication with FTC rulings.

(3) The Chiropractic and the settlement of the Pennsylvania suit brought by a chiropractor against the AMA, the Pennsylvania Medical Society and other organizations.

The decisions made by the House in these regards will have a profound effect on the future course of the AMA's activities. On these issues there were strong feelings on each side, but by the time of the close of the meeting it is hoped that the divisiveness had degenerated and no long term breach would result.

We have attempted to bring you the highlights of this meeting, but should you be interested in more of the proceedings, please refer to the December 15, 1978 Convention Issue of the AMA News which reports all the actions in detail.

This is my last report to you as a delegate to the AMA. It has been an educational experience for me and I am very appreciative of your permitting me to serve. I cannot leave without urging that you participate and become a member of the AMA. It is truly the only national organization through which the voice of medicine can be heard. It is likewise true that oftentimes we are not in agreement with what the AMA may do. Most of our disagreements are based on procedure, not deep philosophy. The AMA is a most democratic organization as is manifest by complete reversal of its position on national health insurance. Input from its members is accepted, considered, and a determination is made by the House as a whole. At a time when the medical profession has so many adversaries, I would encourage your joining the AMA as well as your constituent and state society. To paraphrase an old expression; it is the little differences (procedural) that divide us. In the big issues (quality medical care, unfettered) we are as one. AMA offers to serve as that strong voice and your input is needed. ☐

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OBSTRUCTIVE JAUNDICE CAUSED BY CAVERNOUS TRANSFORMATION OF THE PORTAL VEIN POST NEONATAL OMPHALITIS*

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Extrahepatic portal obstruction developing in the neonatal period usually presents with manifestations of portal hypertension. Obstructive jaundice has not previously been described as a complication of this condition.

CASE REPORT

A twenty-two year old white male presented with jaundice, decreased appetite and tiredness for six months. He had known portal hypertension which followed neonatal omphalitis and portal vein thrombosis. At age four, splenectomy and lieno-renal shunt was done because of bleeding from esophageal varices. Two further self-limiting episodes of gastrointestinal bleeding occurred in his teens.

He was normally developed and showed no other abnormalities apart from jaundice. Total bilirubin was 10.4 with a direct fraction of 7.2. Alkaline phosphatase was elevated to 344 but liver function tests were otherwise normal. Liver biopsy was unsuccessful but was complicated by puncture of the gallbladder with hemorrhage into it, necessitating laparotomy. The wall of the gallbladder was found to contain varices, one of which was oversewn to control bleeding and a cholecystostube was inserted. Serum bilirubin fell to 3.6. Angiography showed esophageal varices, non-patent lieno-renal shunt, thrombosis of the proximal superior mesenteric vein and cavernous transformation of the portal vein, with varices in the region of the common bile duct. (Fig. 1). The patient was re-explored because tube cholecystography revealed marked narrowing and displacement of the distal common bile duct. Varices were found in the region of the common bile duct and also in the gallbladder wall, with no other lesion. Portal pressure was 45 mg. Hg. The gallbladder was judged unsuitable for cholecystojejunostomy and was removed. A T-tube was inserted into the common bile duct and he recovered well, apart from an episode of cholangitis, which responded to antibiotics. Serum bilirubin fell to normal but a T-tube cholangiogram showed persistent narrowing and displacement of the common bile duct by periportal varices, which also showed fine linear calcifications (Fig. 2) Mesocaval shunt and choledochoenterostomy are under consideration.

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DISCUSSION

In the neonate, the umbilical vein forms a potential pathway for the spread of phlebitis and thrombosis from the umbilicus to the portal vein. Described causes of this complication include umbilical sepsis, exchange transfusions, laevulose infusions, dehydration and congenital anomalies of the portal vein.[1, 2] Some cases are idiopathic. Hepatopedal flow from splenic and mesenteric veins to the liver is maintained by the development of collateral venous channels in the lesser omentum, so-called cavernous transformation of the portal vein. (Fig. 1) The inadequacy of these collateral vessels causes portal hypertension and portal-systemic shunting via well described collateral vessels.[2]

The most common presentation of extra-hepatic portal obstruction is bleeding from esophageal varices. Salicylates have been thought to be a contributing factor in some patients.[1] Splenomegaly, hypersplenism, anemia, chronic diarrhea and ascites have also been described.[1, 2, 3] Obstructive jaundice in our patient was caused by compression of the common bile duct by peri-portal varices in the lesser omentum, which were seen at surgery and radiographically. (Fig. 1 and 2)

Calcification in the portal venous system is uncommon and has been associated in all cases with portal vein thrombosis usually complicating cirrhosis. The calcification has occurred in the walls of the portal venous system and within thrombi. The lineal streaks of calcification seen in our patient, (Fig. 2) is an unusual finding and is probably present in the walls of the varices.

SUMMARY

Obstructive jaundice caused by pressure on the common bile duct by varices, in a patient with portal vein thrombosis secondary to neonatal omphalitis, is presented. This is a previously undescribed cause of obstructive jaundice. □

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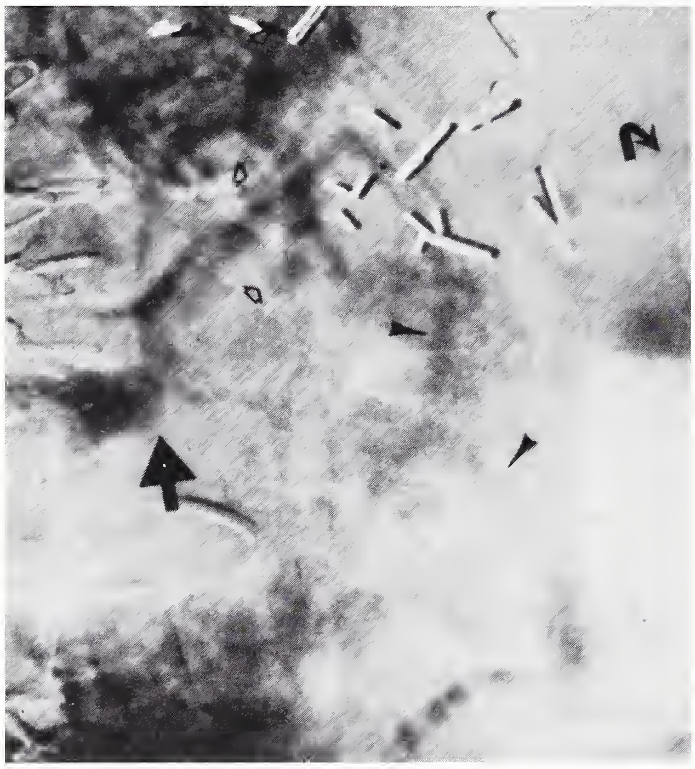

FIGURE 1. Subtraction film of venous phase of superior mesenteric angiogram. Large arrow — Occlusion of proximal superior mesenteric vein. Closed arrow — Peri-portal varices in the region of the common bile duct. Arrow heads — Hepatofugal shunting into coronary and esophageal veins. Curved arrow — filling of intrahepatic portal venous radicles, via peri-portal collaterals.

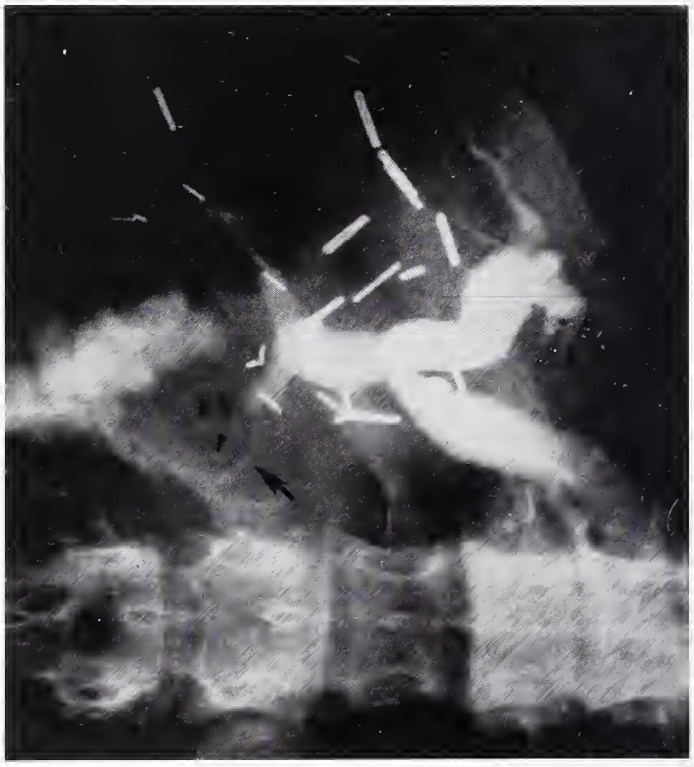

FIGURE 2. T-tube cholangiogram showing displacement and narrowing of the distal common bile duct. Large arrows — Peri-portal varices. Small arrowheads — Linear streaks of calcification in region of varices.

D-PENICILLAMINE IN THE TREATMENT OF RHEUMATOID VASCULITIS

FRANK ADAMS AXSON, M.D., F.A.C.P.*

Rheumatoid vasculitis is one of the more difficult complications of rheumatoid arthritis to treat. D-Penicillamine has been found to be effective in the treatment of this complication in many cases.

CASE REPORT

A 46 year old gentleman was seen in January 1976 with ulcers. The patient had a twelve year history of severe rheumatoid arthritis. The inflammation in his joints was at a peak during the first three years of his disease but remained active with very little improvement in his wrist, MCP and PIP joints, the ankles and knees. He gave an eight or nine year history of rheumatoid nodules and progressive deformity of the hands and feet. He had difficulty walking due to knee involvement for four years and was confined to a wheel chair for three years. He had had recurrent skin ulcers over both lower legs for three years and had undergone skin grafting on two occasions without success. There was also a history of peptic ulcer disease for which he had undergone subtotal gastrectomy in 1973.

Physical examination revealed a thin, chronically ill appearing man who moved very slowly due to pain in his legs. The extremities revealed flexion contractures of the hips and knees. There was ulnar deviation of the fingers and the MCP joints and Heberden's and Bouchard's nodes were present. He had active synovitis with redness, warmth, swelling of the MCP and MTP joints. There were multiple rheumatoid nodules beneath both elbows. The skin showed three cm x two cm deep ulcerations above both medial malleoli. There were erythematous nodules over the chest and hand. The remainder of the examination was within normal limits. His hematocrit was 38 percent, sedimentation rate 34mm per hour, and his white blood count was 4600 with a normal differential. Platelets were adequate. The latex fixation test for rheumatoid factor was strongly positive. The test for antinuclear antibodies was strongly positive with a homogeneous pattern. Urinalysis was normal.

The patient was begun on Ecotrin tabs., Persistin, Motrin, and Cuprimine 250 mgs. tabs, 1 tab. tid. He complained of a "spongy" taste in his mouth. He had a marked improvement in the skin ulcerations from January 1976 to April 1976 at which time the ulcers had decreased to ½ cm in size. The patient discontinued his medication without consultation with me and the ulcers began to enlarge. By May they were 3cm on the right and 4cm over the left ankle. The patient had a maculopapular rash over the neck and buttocks. The patient has not returned since that time.

DISCUSSION

Rheumatoid vasculitis is one of the more difficult complications of rheumatoid arthritis to manage. It may appear as small areas of cutaneous gangrene or ulceration or as a painful nodule lesion on the extremities.[1] At other times it may present as generalized vasculitis with digital ulcerations or deep hemorrhagic blisters with thinning of the skin.[2] At times a diffuse arteritis clinically indistinguishable from polyarteritis appears in patients with rheumatoid arthritis. These cases have been variously interpreted as polyarteritis with early articular inflammation, rheumatoid arthritis followed by polyartheritis (two separate diseases), or as severe active rheumatoid arthritis associated with extensive vascular involvement.[3] Many of these patients are male and are elderly and they frequently exhibit purpura and peripheral neuritis. Most of these patients have rheumatoid subcutaneous nodules and high titers of rheumatoid factor.[3]

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The syndromes of vasculitis tend to be associated with involvement of vessels of different sizes: medium sized arteries, small arteries, and venules. The disease is most frequently described as an arteritis involving blood vessels of all sizes but especially arteries of the vasa nervorum and digital arteries. The inflammation involves all layers of the vessel wall usually lymphocystic predominance but with neutrophils and plasma cells also present.[4] Patients with rheumatoid arthritis and with nodulosis and ulcers tend to have high titers of rheumatoid factor and frequently have hypocomplementemia, cryoglobulinemia and 7 S IgM with greater frequency than the usual patients with rheumatoid arthritis without extra-articular manifestations.[4] Patients with involvement of the medium rather than small arteries tend to have skip lesions associated with fibrosis and intimal proliferation with thrombosis. The third form of vasculitis involving venules may show two cellular patterns. Those patients with serum hypocomplementemia tend to have predominantly neutrophils in cellular infiltrate, whereas lymphocytes predominate in the normocomplementemic group.[4] Venulitis is recognized as erythemateous papules in which the erythema does not blanch with pressure. The venulitis tends to occur in patients with extensive underlying disease and is generally associated with positive rheumatoid factor. Arteritis and venulitis may occur in the same patient.[4] The venulitis may occur in other diseases including Sjogren's syndrome, systemic lupus erythematosus and benign hypergammaglobulinemic purpura.

Bywaters and co-workers have classified rheumatoid vascular lesions into three main pathological groups:

1. Digital arterial narrowing with intimal proliferation but without necrotic or granulomatous lesions;
2. Sub-acute lesions in small blood vessels within muscles;
3. Severe widespread necrotizing arteritis indistinguishable from polyarteritis nodosa.[5]

In a review of rheumatoid vascular disease, clinic presentation included lesions along the nail-edge and petechiae at the fingertips, Reynaud's phenomenon, cutaneous nodules, chronic leg ulcers, erythematous macular and papular rashes, and livedo reticularis.

In 260 hospitalized patients, rheumatoid vasculitis was found in 40 patients. All 40 patients were seropositive. Males made up 32.5% of the patients. This study did not find an excess of vasculitis in patients treated with corticosteroids. The authors felt that the absence of vascular lesions in patients treated with corticosteroid drugs for diseases such as ulcerative colitis made it unlikely that the medications did more than trigger an occult tendency to rheumatoid vascular disease.[5] In some patients, vascular lesions of the fingers and leg ulcers may be precipitated by trauma or cold exposure. Eventually some long surviving patients may develop sudden showers of arteritic lesions even while bedridden. The author found a much poorer prognosis in patients with vascular lesions than in seropositive patients without vascular disease.[5]

A review of 127 hospitalized patients found 76% to have one or more extra-articular complications including nodules, pulmonary fibrosis, digital vasculitis, skin ulceration, lymphadenopathy, noncompressive neuropathy, splenomegaly, episceritis, or pericarditis. Males accounted for 41% of those with one extra-articular manifestation and for 40% of those with two extra-articular manifestations. Males accounted for 23% of those with no extra-articular features of rheumatoid arthritis.[6] The study found a 20% five year mortality in the total group of patients. The mortality was twice as great in the group with extra-articular features, especially in patients aged 50 years or less. The authors found a correlation between the presence of extra-articular features and evidence of persistent articular inflammatory activity, x-ray stages of more severe disease, and the presence of soluble immune complexes and high titers of rheumatoid factor. The extra-articular features did not seem to be related to corticosteroid therapy or the duration of rheumatoid disease.[6] A pathological study of systemic vasculitis found positive rheumatoid factor in 5 of 16 patients with suspected immune complex vasculitis.[7] Vasculitis has been found in patients who have never received corticosteroid therapy.[8]

Penicillamine was discovered by Abraham and his colleagues in the acid hydrolysates of Penicillin in 1942.[9] It is a beta beta dimethylcysteine which is a sulfur containing amino acid. It is absorbed through the intestinal wall with peak blood concentrations one hour after ingestion. It is oxidized by the cytochrome system and excreted as a disulfide.[10] The drug has now been used in the treatment of Wilson's disease, cystinuria, gold and lead poisoning, scleroderma, primary biliary cirrhosis, chronic active hepatitis, muscular dystrophy, and rheumatoid arthritis. The mode of action of penicillamine in rheumatoid arthritis is still unknown. Studies have suggested that its action may be due to inhibition of lysosomal enzyme release in connective tissue membranes. Other studies suggest the effect of inhibition of collagen cross-linking.[9] The drug does not act on macroglobulins. It is not an immunosuppressive drug. The drug inhibits certain viruses in vitro. There is no evidence that the drug's effect in chelating various metals has any relationship to its clinical effect in rheumatoid arthritis.[11]

Penicillamine was first used by Jaffe for rheumatoid arthritis in 1964. Initial doses used were 2 to 3 grams per day. Subsequent studies have used lower doses of penicillamine with less complications.[12] The efficiency of D-penicillamine has been established only for rheumatoid arthritis, seropositive and seronegative, both adult and juvenile. It is not effective in ankylosing spondylitis, psoriatic arthritis, and other B-27 associated variants.[13] At present the drug is used in situations similar to those for chrysotherapy — rheumatoid arthritis with sustained progressive inflammation, not responsive to more conventional therapy. It has been found to be of particular value in treating extra-articular manifestations of rheumatoid arthritis including vasculitis, rheumatoid lung disease, Felty's syndrome, amyloidosis, and rheumatoid nodulosis.[13]

Jaffe currently suggests beginning with 250 mg of D-Penicillamine per day. At three-month intervals, the dose is increased by 250 mg per day until 750 mg to 1 gram per day is reached.[13] In large trials from the United Kingdom, penicillamine has been much better tolerated in smaller doses than the initial 2 to 3 grams. No patient who took penicillamine for 6 months or more left these trials with worse arthritis than on beginning the study. Two-thirds of the patients taking 1200 mg per day and ½ of patients taking 600 mg per day complained of side effects and withdrawals from treatment occurred in 40% and 26% of these groups. Reasons for discontinuing the drug included thrombocytopenia, neutropenia, proteinuria, buccal ulcers, and diarrhea, and abnormalities of taste or rashes.[14] In the 1500 mg group, nine of 21 patients discontinued the drug in the first six months and two more discontinued therapy. Skin vasculitis lesions disappeared after six months of therapy in all nine patients who had the lesions when the penicillamine was begun.[14]

A study comparing gold and D-penicillamine found that 26 (63%) of patients were withdrawn from gold therapy and 21 (45%) withdrawn from Penicillamine. Rashes were the most common cause for stopping gold (61%) whereas rashes (19%), nephrotic syndrome (19%) or lack of effect (24%) were the most common causes for withdrawing penicillamine. Of those patients continuing treatment with penicillamine for two years, 12% had reduction in the size of one or more joint erosions, and 15 (62%) exhibited no, or minimal changes on x-ray. Of those treated with gold, reduction in erosions occurred in two patients (10%) and no or minimal changes in seven (35%).[15] There are no known interactions between D-penicillamine and anti-inflammatory or analgesic drugs.[14] There is no correlation between the toxic manifestations of gold and penicillamine, although two patients who discontinued penicillamine due to oral ulcers had similar ulcers caused by gold, and one patient had albuminuria on both medications. This study found marked improvement in eight patients and moderate improvement in six patients from a group of 17 patients with severe rheumatoid arthritis who were treatment failures on chrysotherapy. There was a 25% incidence of mucocutaneous, renal and hematologic effects requiring discontinuation of the therapy.[16]

A study from Japan found clinical improvement in 65% of patients treated with D-penicillamine and in 27% of the control group. Adverse reactions occurred in 34% of the controls and 49% of the drug group. In the drug group, 27 out of 51 patients with positive rheumatoid factors at the onset had changed to negative after 24 weeks, whereas none of the six patients with negative rheumatoid factor turned to positive.[17]

A study comparing azathioprine and penicillamine in the treatment of rheumatoid arthritis found a significantly greater rise in hemoglobin and fall in sedimentation rate among the patients receiving penicillamine. Ten of 33 patients receiving azathioprine and five of 32 patients receiving penicillamine had to stop treatment due to side effects.[18]

A recent review found an overall complication rate of 53% in 63 patients receiving a total of 83 courses of D-penicillamine. Life-threatening complications occurred in two patients including one case of aplastic anemia and one case of nephrotic cyndrome. An additional patient that was referred with aplastic anemia was discussed. Minor complications included rash in 18%, loss of taste in 16%, dyspepsia in 11%, oral ulceration in 7%, and proteinuria of less than 3 grams per day in 8%.[23] In the patient with aplastic anemia, there was no evidence of thrombocytopenia as an early warning. The authors found an overall complication rate of 69% with cessation of therapy in 39% of courses of therapy. The authors concluded that the toxicity of penicillamine is underestimated.[23] In his recent review, Jaffe suggested that the drug be discontinued when leukopenia of 3,000 WBC's per cubic mm or less occurs or proteinuria exceeds 2 grams per 24 hours. The authors suggested that patients with altered taste perception be continued on the medication and that zinc gluconate be added to hasten the restoration of taste. He pointed out that a history of penicillin allergy is not a contradiction to D-penicillamine. Patients over 60 years of age were found to do less well on D-penicillamine. This paper also discussed the association of D-penicillamine with Goodpasture's syndrome, myasthenia gravis, polymyositis, pemphigus, lupus-like disease, and possibly obliterative bronchiolitis.[13]

A recent case report discussed a patient receiving D-penicillamine who developed signs of polymyositis associated with myocarditis, proteinuria, and hypocomplementemia within one month of beginning therapy.[19] Another report described a patient with myasthenia gravis receiving 1.5 grams of D-penicillamine. The author felt that this complication might be dose-related.[20] A recent review suggested that anti-striational antibody, anti-acetylocholine receptor antibody, and thymic hyperplasia may be independent effects of penicillamine therapy.[21]

A study from England found delayed wound healing in 8 operations of 42 operations involving patients on D-penicillamine. This was due to superficial infection in two cases, wound hematoma in one case, and separation of wound edges in seven cases. The authors compared a 19% incidence of delayed wound healing in patients on penicillamine with a 14.3% incidence of delayed healing in patients on corticosteroids.[22]

SUMMARY

D-penicillamine seems to be quite useful in the treatment of certain complications of rheumatoid arthritis. It would appear that the toxicity of this drug is quite significant and has been underestimated. □

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ANALYSIS OF THE SCMA SURVEY ON CONTINUING MEDICAL EDUCATION

GERALD E. COSTELLO, Ed.D.*

One of the hottest and most debated topics within the medical profession over the past several years has been Continuing Medical Education (CME). As one examines contemporary medical practice and the relationship between CME credits and various privileges associated with medicine, one sadly finds little uniformity among states, specialty societies, national professional organizations and/or state medical associations regarding credit currencies, reporting systems, program accreditation procedures or any number of factors relating to CME. In many states, mandatory CME has become commonplace and is legally tied in with physician licensing, specialty area recertification, specialty society membership, state medical association membership, hospital privileges or other medically related activities. It appears that the positive effects of the once voluntary category credit hour system originally established by the AMA as related to the Physicians' Recognition Award have perhaps become changed to the point where the original intention of the award appears lost, in some states, through legislation as related to medical privileges. Subsequently, the Physicians' Recognition Award and CME activities are viewed negatively by some physicians.

The afore-mentioned points should exist as issues of true concern for physicians especially since to date there is no evidence supporting a strong correlation between physician competence and the number of CME credits earned. There do seem to be, however, strong indications that probabilities do exist linking the two and, therefore, CME and physician involvement and continued support of programs should be encouraged.

The South Carolina Medical Association in responding to a perceived need to begin addressing the CME issue as it relates to South Carolina has authorized the preparation, dissemination and analysis of a questionnaire specifically dealing with those aspects of CME which have been somewhat controversial within the medical community. The express purpose of the questionnaire was twofold. First, it was hoped that the results would be of value to the SCMA Committee on Medical Education whose charge is to be the study, co-ordinating, implementing, liaison and supervisory group with respect to activities of the Association in matters of the continuing education of physicians in South Carolina. Secondly, results of the study could be published in *The Journal* so that the sentiments of South Carolina physicians concerning CME would be known and any subsequent action by the SCMA relative to the topic could be assessed in terms of compliance and compatability with the desires of the responding medical community.

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In December of 1978, the SCMA Committee on Medical Education mailed out a total of 4,065 CME questionnaires. The number included what was thought to be every physician, interns and residents included, in South Carolina. A total of 905 questionnaires (22.3%) were returned for analysis. The Committee feels this was a very respectable response and considers the return to be a valid sampling of the South Carolina medical profession's feelings concerning CME.

An attempt was made in the analysis of the data gained from the questionnaires to separate specific specialty groups, even through not all the specialties responding were coded for analysis. Family practice amassed the greatest number of respondents (15.8%) with surgery being next (13.81%). It should be noted that general practice was treated separately from family practice. (Table I)

The results were also coded relative to type of practice (Table II), size of the primary hospital where the practice was being conducted (Table III) and SCMA membership status. (Table IV)

The first part of the questionnaire, questions 1-3, dealt with the desired nature of CME as either being voluntary or mandatory and, if mandatory, what the desired form of requirements are. Question one is based upon an assumption that at some point physicians will have to demonstrate their being current in terms of medical skills. The question asks what this proof should be based upon. Forty-eight (5.3%) favored periodic examination only, 413 (45.6%) favored participation in CME activities only, 149 (16.4%) favored patient care evaluation only and 389 (42.9%) favored some combination of these. It should be noted that in most of the questions the percentages do not add up to 100% due to either multiple responses or no response at all.

Question 2 asks whether respondents are opposed to or are in favor of mandatory CME. Much to the surprise of many, only 21.6% (196) felt it should be totally voluntary, whereas a vast majority of the respondents favored some degree of mandatory CME. Two hundred and ninety-nine respondents (33.0%) favored approximately 20 hours of CME per year as a requisite for practicing medicine with 343 (37.9%) favoring substantial amounts (150 hours every 3 years) being required to maintain certain privileges associated with the practice of medicine. Sixty-

TABLE I

Breakdown of Respondents by Specialty

	Number of Respondents	Percentage of Total
Family Practice	143	15.80
General Practice	58	6.40
Internal Medicine	82	9.06
Obstetrics-Gynecology	66	7.29
Psychiatry	34	3.75
Pediatrics	51	5.63
Surgery General Orthopaedic and plastic	125	13.81
Urology	23	2.50
Other specialty or response	323	35.68

TABLE II

Breakdown of Respondents By Type of Practice

	Number of Respondents	Percentage of Total
Solo	294	32.48
Institution	173	19.12
Partnership or Group	388	42.87
Not In Practice	31	3.42
No Response	19	2.09

TABLE III

Breakdown of Respondents By Size of Primary Hospital

	Number of Respondents	Percentage of Total
0-100 Beds	104	11.49
101-200 Beds	132	14.58
201-500 Beds	228	25.19
500 plus Beds	313	34.58
No Response	128	14.14

TABLE IV

Breakdown of Respondents By S.C.M.A. Membership Status

	Number of Respondents	Percentage of Total
S.C.M.A. Member	661	73.03
S.C.M.A. Non-Member	166	18.34
Did Not Indicate Membership Status	78	8.61

seven respondents (7.4%) were indifferent with 27 (2.9%) not specifying any preference.

In an attempt to more clearly analyze this point, further computation was done on question 2 with regard to specialty response (Table V) and also SCMA membership status. (Table VI)

TABLE V

Attitude Toward Mandatory CME By Specialty

	Opposed Percent	In Favor Percent	Indifferent Percent
Family Practice	17.48	76.93	3.49
General Practice	27.58	37.92	13.79
Internal Medicine	25.61	68.29	4.87
Obstetrics-Gynecology	30.30	57.57	9.09
Psychiatry	23.52	70.58	2.94
Pediatrics	15.69	72.54	5.88
Surgery	17.60	71.20	6.40
Urology	13.04	65.21	13.04
Other Specialty or No Response	21.76	70.03	8.51

TABLE VI

Attitude Toward Mandatory CME By S.C.M.A. Membership Status

	Opposed Percent	In Favor Percent	Indifferent Percent
S.C.M.A. Member	23.60	65.35	8.61
S.C.M.A. Non-Member	15.06	77.21	4.81

When the physician's specialty is taken into account there appears to be little difference among those coded with the exception of general practice. Family practice has the greatest percentage (76.93) responding favoring some form of mandatory CME with the general practitioners only having 37.92 percent of the respondents favoring some form of mandatory CME. It is interesting to note the general practitioners are the only group with less than half of the respondents favoring some form of mandatory CME. When membership status is considered as a possible variable, there appears to be some difference between SCMA members and non-members, 65.35% to 77.21%.

Question 3 was stated in conditional terms that "if CME is to be mandatory, I would prefer that it be tied in with?" Because of the nature of the question, multiple answers were common. The results of this question can be found in Table VII with responses listed in order of preference. For purposes of additional clarity and information, the preferred vehicles for mandatory CME by specialty are listed by percentages of response in Table VIII. Review of this table seems to indicate that respondents are supporting what already exists in many instances as a requirement within their particular specialty. Psychiatrists favor strongly CME as a requirement for membership

TABLE VII

Preferred Vehicle for Mandatory CME

	Number of Responses	Percentage of Total
Periodic Re-certification in My specialty Area	307	33.90
Periodic Re-registration of My License to Practice Medicine	222	24.50
Membership In My Specialty Society	184	20.30
Hospital Staff Privileges	173	19.10
Membership in the South Carolina Medical Association	111	12.20
Peer Review	100	11.00
No Preference	64	7.00
Other Or Did Not Specify	28	3.00

TABLE VIII

Preferred Vehicle for Mandatory CME By Specialty

	Family Practice	Pediatrics	OB-GYN	Surgery	Psychiatry	Internal Medicine	General Practice	Urology	Other
Medical License	18.18	29.41	19.69	21.60	32.35	31.70	24.13	26.08	26.18
S.C.M.A. Membership	18.88	7.84	9.09	11.20	11.76	9.75	22.41	8.69	10.41
Specialty Re-Certification	51.47	43.13	45.45	24.80	20.58	21.95	5.17	30.43	35.64
Specialty Society Membership	22.37	5.88	12.12	30.40	44.11	12.19	3.44	26.08	21.76
Hospital Staff Privileges	18.88	17.64	18.18	23.20	20.58	21.95	13.79	21.73	18.29
Peer Review	5.59	9.08	13.63	12.00	8.82	10.97	20.69	17.39	10.41
No Preference	3.49	9.80	7.57	6.40	2.94	10.97	15.51	8.69	6.30
Other	2.09	1.96	3.03	1.60	0	1.22	6.89	4.34	4.41

in their specialty society as do some surgeons. This is consistent with what is already the case for the psychiatrist as well as the neuro, abdominal and plastic surgeons. Family physicians, Pediatricians and Obstretrician-Gynecologists strongly favor CME for specialty recertification. Responses from the general practitioners seem to be varied as to their feeling concerning CME with no vehicle strongly preferred.

Most of the remaining parts of the questionnaire dealt with the nature of CME programming in terms of places held, mechanisms for planning CME topics, methodologies employed, program appropriateness and program length. These shall be discussed separately in another paper in that they seem to support a different issue. Question number seven however did ask "what role(s), if any, do you believe the South Carolina Medical Association should play in the CME area?" The results of this question can be found in Table IX with responses listed in order of preference. Review of this table indicates strong preference for the SCMA to become actively involved in this area with its assuming an overall leadership role being most strongly supported.

DISCUSSION

Perhaps the most important result of the survey was finding out that clearly the majority of physicians responding do favor some degree of documentation of continuing medical education. Some might say that only those physicians strongly favoring CME responded to the questionnaire to which one could reply by stating that those opposed to CME had the same opportunity to respond and therefore, the results represent a valid sampling of S. C. physicians' opinions on the topic. Related to this point and perhaps even more crucial is the question, if CME should become mandatory, with what mechanism does the CME become associated. The survey indicated on the whole that recertification in ones' specialty and medical relicensing are most desirable with membership in the state medical association and maintaining hospital privileges being considerably less popular.

The Committee on Medical Education feels that the results of the survey will be of considerable value in the subsequent development of its plan for CME in South Carolina. The time, effort, money and manpower used to develop, disseminate and analyze the questionnaire appears to be well spent and most worthwhile. At this point in time South Carolina is one of only about a dozen states which has not already mandated, or are in the process of legislating CME as a requirement for relicensure or membership in the state medical associations or societies. It is felt that this perhaps is good but it also indicates that the medical profession needs to seriously consider the CME issue and how it can be addressed in the future most effectively, efficiently and most palatably. ☐

TABLE IX

Breakdown Of Responses To The Role The S.C.M.A. Should Assume In The CME Area

	Number of Responses	Percentage of Total
Should Serve As The Main Co-ordination Agency For CME Programs Receiving Category I Credit in S. C.	317	35.02
Should Maintain M.D. Records Concerning CME Credits	242	26.70
Should Closely Supervise The Cost of CME Programs Offered in S. C.	217	23.90
Should Assume No Role Greater Than Any Other Agency Or Organization in S. C. Relative to CME	207	22.80
Should Itself Be A Provider of CME	184	20.30
Should Oversee Advertising of CME	176	19.40
Should Assume A Lesser Leadership Role Concerning CME in S. C.	36	3.90
No Response Or Other	50	5.50

MALPRACTICE: AN ANALYSIS OF CLAIMS AGAINST THE SOUTH CAROLINA JOINT UNDERWRITING ASSOCIATION

BOYCE M. LAWTON, JR., M.D.*

From March 1976 to October 1978, a total of 153 claims were made against providers of medical service in South Carolina. Types of claims, the medical specialties involved and illustrations follow. During this same period, 205 claims were made against hospitals; however, an analysis of hospital claims is not included in this report.

Specialties involved are shown in Table 1. Family practitioners and general practitioners combined represent 16.9 percent. All the surgical modalities (including orthopedic surgery and urology) represent 43.1 percent. As a single group, however, the OB-GYN specialty had the highest percentage of claims with 15 percent.

The type of problem causing a claim is illustrated in Table 2. Twenty-one of these claims represent deaths, some of which involved less than optimal care. Ten claims were the result of foreign bodies retained after surgery — with sponges responsible for seven of these. A broad category involving errors of omission or commission include the following groups: inadequate care — patient expired, complications of surgery, improper care or treatment, missed diagnosis, negligence, foreign bodies retained, and incorrect medication. These account for 49 percent of all claims.

Nineteen of these claims (12.4 percent), were felt to involve no liability or wrong doing on the physician's part. This does not mean no payment was made.

Specific events that prompted these claims are itemized. Only a brief summary will be given so no patient nor physician can be identified. Under the heading "Inadequate care — patient expired" are:

a) Oxytocic given — neither M.D. nor nurse present at delivery, infant expired.
b) Improperly intubated for 5 minutes — expired.
c) infant expired with epiglottitis — tracheostomy was questionably done too late.
d) Patient fell off stretcher in Emergency Room, transferred to jail and died.
e) Breech, delay in C-Section — stillborn.
f) Delay in referring breech for C-Section — stillborn.
g) Improperly intubated (esophagus) — expired.
h) Child with emesis died of aspiration of vomitus — found to have volvulus — delay in x-ray alleged.
i) Young diabetic delivered stillborn then died herself in diabetic coma.

Complications of surgery included:

a) Carotid artery accidentally severed, patient sustained cerebral thrombosis.
b) Premolars loosened.
c) Scarring of leg from cast.
d) Tooth dislodged during abdominal surgery.
e) Sutures allegedly removed early — wound separated.
f) Brain stem herniation, death after arteriogram.

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TABLE 1

Malpractice Claims Against Providers By Specialty From March 1976 to October 1978

Rank		Claims
1	OB-GYN	23
2	Surgery — General	17
3	Family Practice	16
4	ORS	11
5	GP	10
6	Urology	9
7	Anesthesiologist	7
8	Emergency Medicine	6
9	Ophthamology	5
10	Otolaryngology	5
11	Anesthetist — regular	4
12	Internal Medicine	4
13	Surgery — thoracic	4
14	EENT	3
15	Radiology	3
16	Pediatrics	2
17	Surgery — Cardiovascular	1
18	Dermatology	1
19	Pathology	1
20	Gynocology	1
21	Neurosurgery	1
22	PS	1
23	DDS	1
24	Chiropractor	1
25	Psychology	1
26	Designation not established	15

g) Facial paralysis after open reduction of fractured mandible.
h) Scalp injury, caused undetermined, during breast surgery.
i) Pneumothorax after intercostal nerve block.

Twenty-three claims (15 percent) were felt to be the result of negligence. There were three deaths in this group — and include:

a) Uterus perforated during D & C — (2).
b) Ureter ligated during hysterectomy — (1); Oophrectomy — (2); Herniorrhapy — (1).
c) Urinary bladder lacerated with subsequent vesicovaginal fistula during hysterectomy — (4); C-Section — (2).
d) Patient treated for PID, died of ruptured uterus with 4-5 month fetus. No lab work.
e) Intubated in stomach for circumcision — expired.
f) Youth circumcised by cauterization, shaft of penis lost.
g) Still birth — alleged due to failure to deliver after gestation date.
h) Tubal ligation unsuccessful — patient became pregnant.
i) Chiropractor fractured rib of patient during adjustment.

TABLE 2

Causes of Malpractice Claims Against Providers From March 1976 to October 1978

Problem Causing Claim	**Number of Claims**
Negligence	23
No liability seen — questionably frivolous	19
Improper treatment/care	17
Missed diagnosis	16
Complications of surgery	13
Foreign body retained after surgery: sponge — 7; surgical needle — 2; piece of stapler instrument — 1	10
Inadequate care — patient expired	10
Reaction to medication: rash — 1; Penicillin treatment — 1; Brain damage and blindness — 1; Steven Johnson Syndrome — 1; hemoptysis — 1	7
Information insufficient to state cause	5
Unsatisfactory results from surgery	5
Cardiac arrest during surgery — expired	4
Complications after injection	4
Cardiac arrest during surgery — revived	3
Incorrect medication or given incorrectly	3
Complications of treatment — eardrum ruptured while cleaning ear — 1; keratitis — 1; IUD (Dalton Shield necessitated hysterectomy) — 1	3
Injury due to equipment defect	2
Fall, in or leaving office, resulting in injury	2
Fraud alleged — re surgery performed	1
Sued for bill — already paid	1
Invasive procedure resulting in death — arteriogram	1
Inadequate or no consent for procedure	1
Surgery performed but not indicated	1
Complication of cannulization	1
Drug reaction resulting in death	1

Improper treatment categorizes another group of claims:

a) Ampicillin given when known allergic to penicillin — probably permanent neurological deficit.
b) Failed to diagnose piece of metal in eye — no x-rays.
c) Hand injury improperly treated — should have secured surgical consultation or referred patient.
d) Torsion of testicle — delay in treating youth with only one testicle.
e) Term pregnancy, false labor was diagnosed for two days — delivered stillborn third day.
f) Fibroids suspected — referred to surgeon who did laporotomy — patient pregnant — no pregnancy test done.
g) Delay alleged in seeing footling breech — brain damage.
h) Laceration of hand, flexor tendon not repaired.

i) Dalkon shield not removed as requested when tubal ligation done — developed infection from IUD later.

Missed diagnoses include:

a) Myocardial infarction not diagnosed for five days.
b) Failed to diagnose ectopic pregnancy, no lab work, patient died.
c) Osteogenic sarcoma with metastasis not diagnosed nor treated for two months.
d) Ruptured appendix diagnosed as PID, unnecessary hysterectomy, salpingoophorectomy.
e) Periappendiceal abscess diagnosed and treated as fecal impaction.
f) Ruptured appendix diagnosed and treated as gastroenteritis.
g) Patient died of hemorrhagic pancreatitis — not diagnosed.

Complications after injections of medication were:

a) Neuropathy of dominant arm after injection.
b) Abscess of buttock requiring incision and draining.
c) Radial nerve damaged after Phenergan injection.
d) After IM injection of Tetracycline, teenager had syncopal episode outside MD's office — small laceration.
e) Penicillin given IM to patient with two previous episodes of urticaria resulting in death of patient.

Claims classified as without merit or frivolous include:

a) Surgeon repaired improper surgery done by another, but was included in suit.
b) Spontaneous abortion attended by first MD — bleeding persisted. Seen by second MD who removed remainder of placenta. Second MD sued for not removing all placenta at time of abortion.
c) Male admitted to hospital for elective repair of hernia. Because of excessive demands for pain medication, patient discharged without surgery. Sued for failure to do surgery.
d) Stillborn birth of twins. All treatment was proper within accepted standards of care.
e) Improper care alleged for broken arm — patient did not follow instructions — care was proper.
f) Superficial corneal abrasion alleged after awakening from surgery.
g) Torsion of testicle in youth repaired promptly by surgeon after referral. Surgeon sued for loss of testicle. Referring MD waited two days before referring, however.
h) Back injury alleged due to spinal earlier for C-Section.
i) Claimant alleged antibiotic should have been used and hospital discharge premature — care apparently proper.
j) Sued for sudden infant death — no improper action on physician's part. □

Come Help Us Celebrate The Child

During the current International Year of the Child, St. Jude Children's Research Hospital continues its desperate search for life-saving knowledge about catastrophic childhood disease.

As the only research center devoted entirely to the conquest of the illnesses that destroy the lives of too many children the world over, St. Jude Hospital celebrates the child with every new finding. And there have been so many.

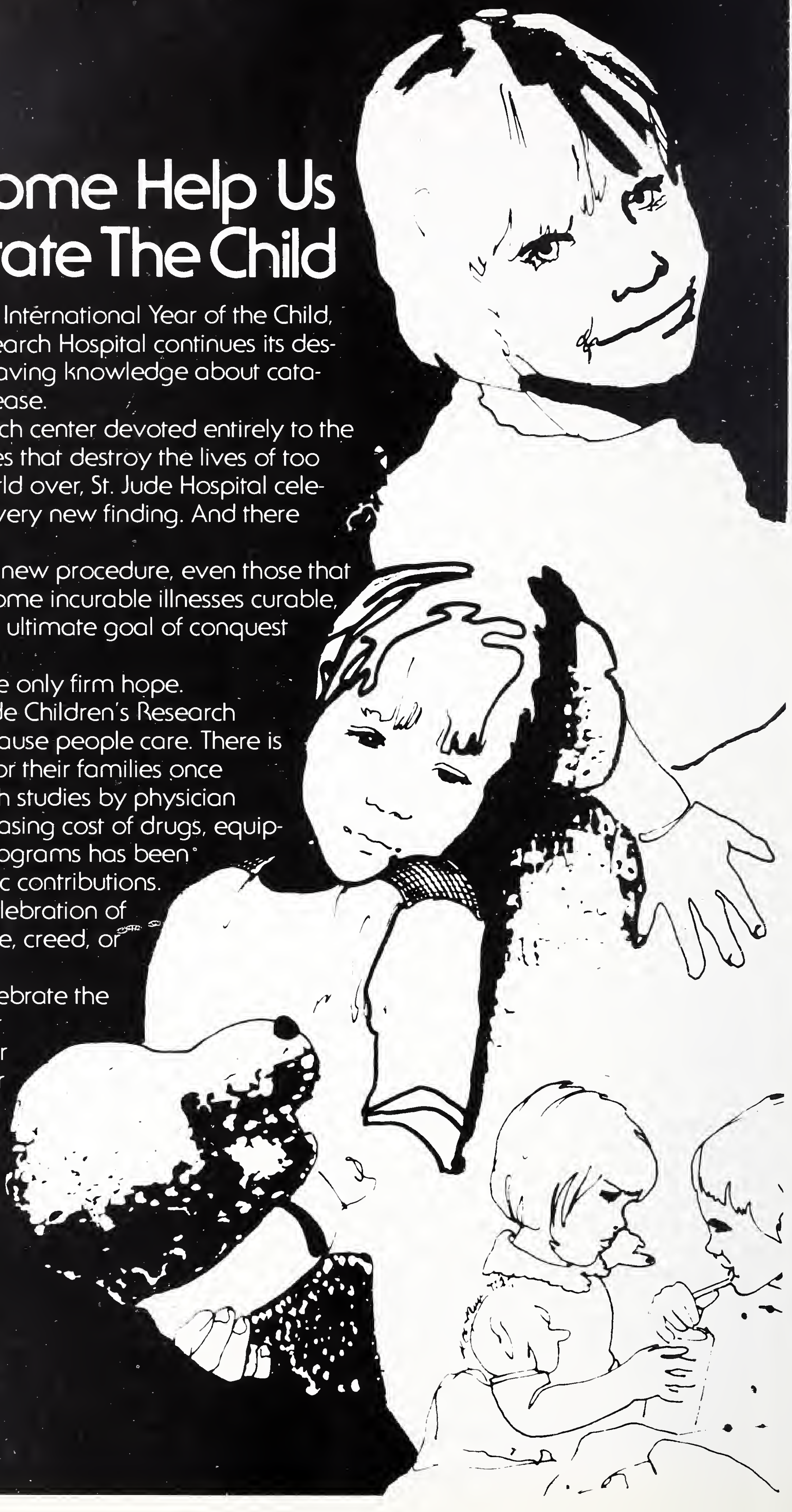
But each priceless new procedure, even those that have already made some incurable illnesses curable, shows how elusive the ultimate goal of conquest remains.

Research offers the only firm hope.

Research at St. Jude Children's Research Hospital continues because people care. There is no charge to patients or their families once admitted to its research studies by physician referral. The ever increasing cost of drugs, equipment, and research programs has been met primarily by public contributions.

This miraculous celebration of the child knows no race, creed, or financial distinction.

Come, help us celebrate the child. Please send your tax-deductible check or your request for further information to St. Jude Children's Research Hospital, 539 Lane Avenue, Memphis, Tennessee 38105.



ST. JUDE CHILDREN'S
RESEARCH HOSPITAL
Danny Thomas, Founder

Editorials

CONTINUING EDUCATION IN SOUTH CAROLINA

In this issue of *The Journal* is published a report by Dr. Gerald Costello summarizing the results of a questionnaire sent to South Carolina Physicians regarding Continuing Medical Education. The Committee on Medical Education of the South Carolina Medical Association in an attempt to determine what the desires and needs are as expressed by physicians in this state distributed to every physician a questionnaire which we hope will provide some guidelines as to future direction as we face this rapidly expanding area of medical interest.

When the American Medical Association first instituted the Physician's Recognition Award and identified the Category I credits as being the prime factor in obtaining this Award, it was little suspected that a formal explosion in Continuing Medical Education would result. What was begun as a program to measure physicians' medical education is evolving into a learning system that can be manipulated legally, economically, and politically. Along with opportunities for learning has come, in some states, legislative restriction and self imposed regulation based on the premise that every doctor must maintain a minimum standard of medical education in order to qualify and to maintain his standards as a practicing physician. There has been much backlash in these areas and perhaps the bad has outweighed the good as we physicians watch yet one more external force being imposed upon us.

We must guard carefully against any such process that would capitalize on a physician's learning experience. Those physicians who belong to Medical Specialty Groups and maintain educational credentials need not address the issue of CME, since their educational requirements are already being met. Many physicians, however, not included in this group welcome some form of system whereby their educational efforts may be calibrated and credited.

An AD HOC Committee appointed by the Committee on Medical Education of the South Carolina Medical Association is in the process of outlining a five year plan for the future of Continuing Medical Education in our State. Much of what is said and done in that Committee is being guided by the data obtained from the questionnaire submitted to State Physicians. I would encourage your careful review of the data presented in this issue of *The Journal* and thereby gain some insight of direction that respondent physicians have presented. At this point, the figures speak for themselves, representing return of 905 questionnaires out of a total of 4,065 sent out. Two issues are imperative: first that we as a state medical association begin to move in some manner as to our formal involvement in the area of Continuing Medical Education; and secondly, that the direction in which we move represents the sentiments and desires of the majority of the physicians of our State.

We owe much to Dr. Vince Moseley who over the past several years has pioneered and spearheaded the CME effort in South Carolina. It was under his direction that we have promoted new and innovative methods of learning now being used. We must continue to experiment in these areas as we pursue better methods for medical learning.

N. B. Baroody, Jr., M.D., Chairman,
SCMA Committee on Medical Education

MALPRACTICE: THE VALUE OF DATA

Gone but not forgotten is the "malpractice crisis" of 1975. The outcome parallels, in some way, that of the "oil crisis" — we grudgingly accept the high costs because we need the services; we regret that no alternatives were found.

In 1975, thoughtful people concluded that the root of the crisis lay not in doctors, lawyers, patients, or insurance companies, but rather in the entire concept of the tort liability system buffered by insurance carriers. The old English concept of tort liability (responsibility for wrongdoing) had somehow become perverted. The transfer of fiscal responsibility to a large group of *potential* wrongdoers created a system in which individuals sought a pot of gold, placed there at no one person's expense. Ultimately, however, the public paid for the pot of gold, and whether it could continue to afford this luxury appeared questionable, at best.

Several alternatives were offered. *Screening panels* could reduce the number of suits filed without just cause, and provide patients with possible claims the benefit of expert testimony without the burden of great expense. *Binding arbitration* could reduce the costs of the litigation process and offer settlement in a calmer atmosphere than that of the courtroom. *No-fault insurance* could provide a far-reaching solution, possibly one in which the group of potentially-injured would assume fiscal responsibility for damages. None of these options gained widespread acceptance.

The American Bar Association formed a Commission on Medical Professional Liability, which included eight lawyers, three physicians, three insurance representatives, and one state social service agency representative. The commission weighed all of the options and concluded that the present system remained the best, despite its limitations. It was pointed out that a major virtue of the present system was its *deterrence function.*[1]

Thoughtful people continue to debate alternatives. One analysis, published last year, argued that the "present system" was *not* necessarily more expensive than the proposed options when viewed from the economic analysis of cost of preventing mishaps. The authors assumed that "the malpractice system exists to discipline the occasional physician who does not (or can not) protect his patients."[2] This assumption is itself debatable. An editorial comment argued that such a "cost-benefit approach is wholly unsuitable" for evaluation of medical malpractice cases, concluding that there remains a need for "sober reassessment of the limits of all coercive technics."[3]

In this issue of *The Journal,* Dr. Boyce M. Lawton provides an analysis of claims made against the Joint Underwriting Association in South Carolina from March 1976 to October 1978. If malpractice claims are to have any widespread deterrence function, we certainly need to see the data; thus, such data are invaluable in demonstrating to us the *specific* types of problems which are likely to result in claims. The judgement that many must pay for the potential wrongdoing of a few, and that an inefficient system which rewards the wronged with only a fraction of its monies should continue, seems harsh. The need for alternatives continues, but meanwhile we can best profit from the "deterrence function" of the present system *only* by our serious attention to data such as those provided in Dr. Lawton's summary.

— CSB

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1. *1977 Report of The Commission on Medical Professional Liability* (Chicago: The American Bar Association, 1977).
2. Schwartz WB, Komesar NK: Doctors, damages and deterrence: an economic view of medical malpractice. New Engl J Med 298: 1282-1289, 1978.
3. Epstein RA: The limits of medical malpractice. New Engl J Med 298: 1311-1312, 1978.

President's Pages

IN UNITY THERE IS STRENGTH

To be President of the South Carolina Medical Association is a great honor. With this honor comes responsibility to the entire membership, individually and collectively. I pledge to you my very best effort to serve you and to represent you.

With thanks to the leadership and farsightedness of Council, Past Presidents, and to the membership that gave the extra financial support when it was needed, this organization has grown and is now stable. It is financially sound and has assembled an excellent staff that attend to the day-to-day affairs with efficiency.

I have two goals in mind for this year that will serve to help us guide our course:

1. *Service to the membership.*
2. *More membership involvement.*

This organization has lofty, long term ideals and objectives which are admirable, appropriate and attainable. To accomplish these, we must establish priorities. The short term priority of this organization is not simply to distribute Newsletters, hold conventions, and publish a *Journal*, but to help you *solve problems.* Problem-solving is a "we" effort; that means the utilization of more members in the decision making process. This, hopefully, can be attained by the improved Commission/Committee organizational structure.

One of my first obligations is, with the advice and consent of Council, to make committee and commission appointments. I urge, if you have not already done so, that you complete the questionnaire that was enclosed with your membership card with regard to your offer of service on the committee that best represents your interest and expertise.

Because medical interests are so diverse, involvement is more than being a member of SCMA. It means a commitment to participate in specialty, county and national medical organizations; a commitment to study the issues that confront us today; a commitment to serve as a good citizen to help influence legislation that involves the delivery of quality medical care. You are the expert — let your thoughts be known.

When the issues are studied, even though our medical interests vary, we will find that we have common problems with common adversaries. SCMA wants to serve as your problem-solver. It can be done by unity that brings strength and success.

"It is the little things of life that create the differences. In the big things of life, we are as one." Let us join together to be the umbrella organization that gives us strength.

Harrison L. Peeples, M.D.
President

The Maker

Examining a Few Myths About Prescribing.

Increasing pressure is being put on the practicing physician to prescribe drugs generically. You are told that brand-name products are universally "expensive" and generic versions are relatively "cheap." To make this case, the most extreme (rather than typical) price differentials are cited. Thus, consumers are led to believe that such differentials are commonplace. Even your knowledge and your motives as a physician are questioned.

Understandably, these views have created myths. We think it's time to examine them in the light of all the facts and ramifications.

MYTH: There are no differences in quality and performance between brand-name products and their generic counterparts. The corollary is that there are no differences among products made by high-technology, quality-conscious, research-based companies and those made by commodity-type suppliers.

FACT: The Food and Drug Administration does a good job in monitoring a generally excellent drug supply. Still, it has nowhere near the resources to guarantee the quality and bioavailability of all marketed products at any given time. Just a few months ago, for example, it noted that batches of tetracycline HCl capsules which met official monograph requirements were not bioequivalent to a reference product. As you know, there is substantial literature on this subject affecting many drugs, including such antibiotics as tetracycline and erythromycin. The record on drug recalls and court actions affirms strongly that there are differences among pharmaceutical companies and their products. Research-intensive companies have far better records than those that do no research and may practice minimum quality assurance.

MYTH: Industry favors only "expensive" brand names and denigrates all generics.

FACT: PMA companies make 90 to 95 percent of the drug supply, including, therefore, most of the generics. Drug nomenclature is not the important point; it's the competence of the manufacturer and the integrity of the product that count.

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JOHN JAMES AUDUBON AND HIS CHARLESTON PHYSICIAN-FRIENDS*

GEORGE E. GIFFORD, JR., M.D.**

In 1831, John James Audubon, George Lehman, and Henry Ward came to Charleston, S. C., to collect birds for Audubon's great *Birds of America*. They were taken in by the Reverend John Bachman, minister of St. John's Lutheran Church and friend of the naturalists and physicians of Charleston.

One of Audubon's biographers describes this association:

> Audubon could have imagined nothing better than this chance meeting with a great-hearted man of science, one not seeking personal glory, but only eager to be of assistance. Blue-eyed, affable, his weathered cheeks deeply dimpled as if by inveterate smiling, Bachman possessed a sense of humor, penetrating mind, and good but plain manners that made their encounter seem luck itself. The Lutheran minister looked the part of a descendant of one of Penn's Rhineland agents. His account of a boyhood visit to the Oneidas was enthralling . . . Tuberculosis in young manhood had compelled the native of Rhineback, New York, to leave a post in Pennsylvania for the milder Charleston.
>
> Bachman's white mansion, set in brilliant gardens, faced the cooling sea breeze. The house stood among roses, jasmine, and mossy green islands bordered by verbena, sweet alyssum, mignonette, and snow-on-the-mountain. Orange wall flowers, wisteria, patches of yucca, splashes of petunias and arborvitae, camellias, and boughs of pomegranate abounded. Trumpet vines, dear to the hummingbird, climbed the magnolias. A kitchen garden was fringed by mulberry and fig trees. Beyond lay a duck pond, a chicken house, pigeon cotes, bee hives, an aviary, and finally, slave quarters.
>
> In the low-ceilinged rooms were formal fireplaces, deep window seats, high wainscoting, Adam furniture, and bits of Dresden china. The gracious Mrs. Bachman, granddaughter of an American Revolutionary hero, seemed accustomed to a flow of guests. Miss Maria Martin, her gifted sister, showed Audubon drawings she had made for Bachman's scientific papers. . . .
>
> . . . he was soon rejoicing, "Bachman takes us all over the country, after specimens, in two carriages."

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* Delivered to the First Annual Meeting of the Waring Library Society, Charleston, S. C., March 9, 1979.

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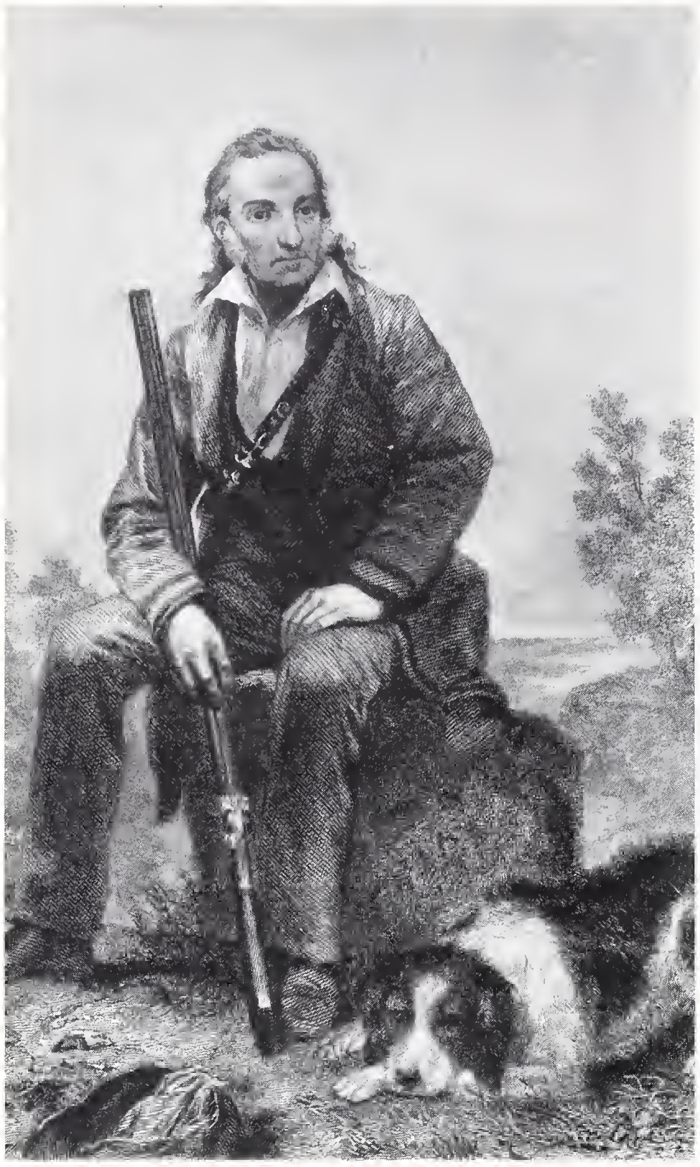

FIGURE 1. John James Audubon.

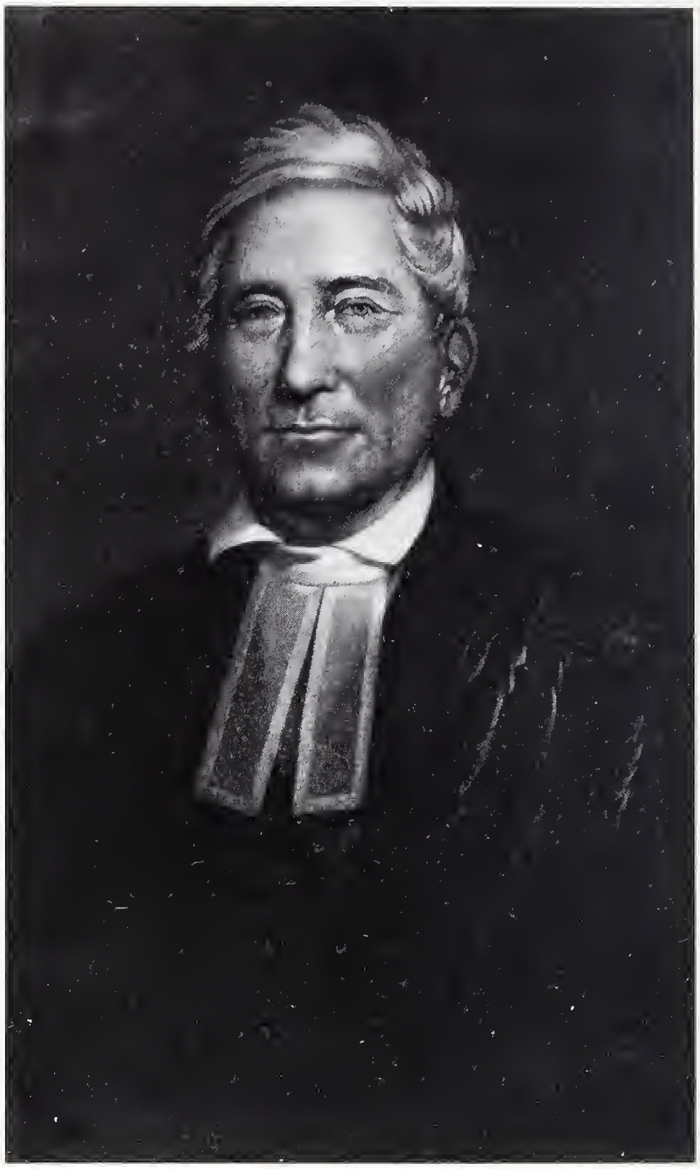

FIGURE 2. The Reverend John Bachman.

> . . . At night the two bosom companions, Bachman and Audubon, wrote and studied in a library den amid stuffed owls and monkeys, atlases, books, bottled reptiles and freaks of nature, caged wild birds, and lively pet rodents.[1]

Bachman later wrote: "We were engaged in talking about Ornithology, in collecting birds, in seeing them prepared, and in laying plans for the accomplishment of that great work which he has undertaken . . . there seems quite a blank in my house since he has gone, for we looked on him as one of our family. He taught my sister [actually sister-in-law] Maria to draw birds; and she has now such a passion for it, that whilst I am writing, she is drawing a Bittern, put up for her at daylight by Mr. Audubon."[2]

The Bachman-Audubon friendship culminated in a family relationship. Two of Bachman's daughters would later marry Audubon's two sons; Mary Eliza Bachman married Victor Gifford Audubon and Maria Rebecca Bachman married John Woodhouse Audubon.[1]

Bachman's friends became Audubon's supporters, for example, the two Drs. Gibbes, Dr. Samuel Wilson, Dr. Leitner, Dr. Strobel, and Dr. E. Ravenel.

> Robert Wilson Gibbes (1809-1866), who practiced in Columbia, S. C., had acquired a large and valuable collection of fossils, paleontology, geology, mineralogy, and conchology. He befriended Audubon, who called him a man of "taste and talent". Lewis R. Gibbes provided assistance in supplying Latin descriptions for the quadrupeds.
>
> Lewis R. Gibbes (1810-1894) received his M.D. from the Medical College of South Carolina in 1836, having published *A Catalogue of the Phaenogamous Plants of Columbia, S. C. and Its Vicinity* in 1835.

> He wrote on astronomy, mechanics, physics, and chemisty and was president of the Elliott Society of Natural History for 37 years (1853-1889).
>
> Dr. Samuel Wilson, Bachman's friend and physician . . . befriended Audubon . . . and supplied him with specimens for *The Viviparous Quadrupeds*. His son, Dr. Samuel Wilson, Jr., became interested in ornithology after his return from medical school.[3]

Dr. Edward Frederick Leitner (1811-1838), who had published a *Botanical Chart* in Charleston, collected specimens for Bachman and Audubon in the Florida Keys. Audubon honored him by including a plant which Leitner had collected in the plate of the Whistling Swan (Plate No. 411) (Figure 3).

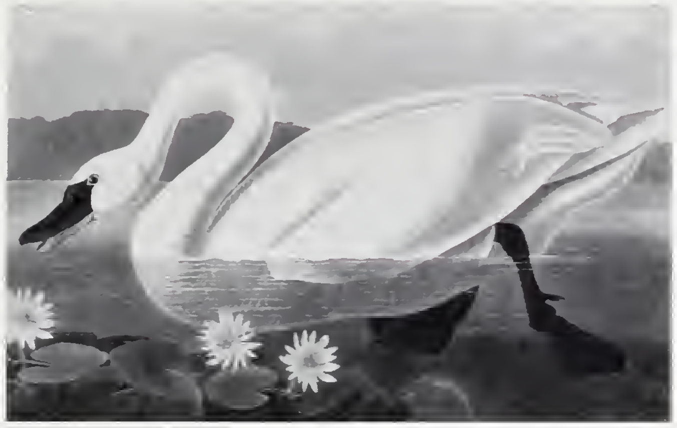

FIGURE 3. A plant collected by Dr. Frederick Leitner, who had published a *Botanical Chart*, appears in this Audubon painting of the Whistling Swan.

> Audubon probably painted this swan in London in 1838. In Havell's engraving, there are three yellow water-lilies of considerable interest. Audubon first referred to the flowers in a letter written from London to John Bachman on April 14, 1838: "Has Leitner published the New Plants he discovered in the Floridas? I ask this latter question because on the 83 number of my work, Plate 411 (the Havell plate number), I have represented a New Nymphea, which unpublished by him, I should like in my letter press to name after Doc^r Leitner's name, 'Nymphea Leitneria'." Edward F. Leitner, the German botanist to whom Audubon refers, had been killed in Florida by Seminole Indians three months before this letter was written. The yellow water-lily came to be considered another extravagant figment of Audubon's imagination, but in 1876 it was rediscovered and today is known as *Nymphaea mexicana*.[4]
>
> Dr. B. B. Strobel, a former Charlestonian who was town physician of Key West, sent shells, bird skins, and tropical plants to Bachman and Audubon and hosted Audubon in Key West.[5]

Dr. Edmund Ravenel (1797-1871) was a physician who held the chair of chemistry and pharmacy at the Medical College and was dean of that institution. Dr. Ravenel was an authority on conchology and published a catalogue of shells in 1834 which was said to be the first of its kind in America. He was active in the Elliott Society of Natural History.[6] Ten of thirteen curators of the organization were physicians; it was characteristic of American science at that time for physicians to be the leaders in the study of natural history.

Undoubtedly, on this visit to Charleston, Audubon and Lehman met the great Charleston physician-naturalist, John Edwards Holbrook (1796-1872) who was Professor of Anatomy at the Medical College of the State of South Carolina at the time of that visit.[7] Lehman executed some of the plates for Holbrook's *North American Herpetology* published in 1842 in Philadelphia. Bachman's sister-in-law, Maria Martin, contributed several drawings and Holbrook made the following acknowledgement, "To Miss Martin, of Charleston, my thanks are also due for some very accurate and very spirited drawings of Carolina Reptiles."[8]

Audubon wrote of his Charleston friends: "It is impossible to do justice to the generous feeling of the Charlestonians, or to their extreme kindness towards me. Many of the gentlemen took the greatest interest in my pursuits; one, Dr. Wilson, presented me with an excellent Newfoundland dog, and other valuable memorials of his regard. Another, Dr. Ravenel, gave me a collection of shells from the adjacent waters."[9]

Audubon painted many birds around Charleston, South Carolina, and included landscape vignettes in several of these. During his first trip to Charleston, he made an expedition to Cole's Island,[10] an account of which is included in his *Ornithological Biography* dated November 10, 1831.

> The Long-billed curlew spends the day in the sea-marshes, from which it returns at the approach of night, to the sandy beaches of the sea-shores, where it rests until dawn. As the sun sinks beneath the horizon, the Curlews rise from their feeding-grounds in small parties, seldom exceeding fifteen or twenty, and more usually composed of only five or six individuals. The flocks enlarge, however, as they proceed, and in the course of an hour or so the number of birds that collect in the place selected for their nightly retreat, sometimes amounts to several thousands. As it was my good fortune to witness their arrivals and departures, in the company of my friend *Bachman*, I will describe them.
>
> Accompanied by several friends, I left Charleston one beautiful morning, the 10th of November 1831, with a view to visit Cole's Island, about twenty miles distant. Our crew was good, and although our pilot knew but little of the cuttings in and out of the numerous inlets and channels in our way, we reached the island about noon. After shooting various birds, examining the island, and depositing our provisions in a small summer habitation then untenanted, we separated; some of the servants went off to fish, others to gather oysters, and the gunners placed themselves in readiness for the arrival of the Curlews. The sun at length sunk beneath the water-line that here formed the horizon; and we saw the birds making their first appearance. They were in small parties of two, three, or five, and by no means shy. These seemed to be the birds which we had observed near the salt-marshes, as we were on our way. As the twilight became darker the number of Curlews increased, and the flocks approached in quicker succession, until they appeared to form a continuous procession, moving not in lines, one after another, but in an extended mass, and with considerable regularity, at a height of not more than thirty yards, the individuals being a few feet apart. Not a single note or cry was heard as they advanced. They moved for ten or more yards with regular flappings, and then sailed for a few seconds, as is invariably the mode of flight of this species, their long bills and legs stretched out to their full extent. They flew directly towards their place of rest, called the "Bird Banks", and were seen to alight without performing any of the evolutions which they exhibit when at their feeding-places, for they had not been disturbed that season. . . .
>
> . . . Now, Reader, allow me to say a few words respecting our lodgings. Fish, fowl, and oysters had been procured in abundance; and besides these delicacies, we had taken with us from Charleston some steaks of beef, and a sufficiency of good beverage. But we had no cook, save your humble servant. A blazing fire warmed and lighted our only apartment. The oysters and fish were thrown on the hot embers; the steaks we stuck on sticks in front of them; and ere long everyone felt perfectly contented. It is true we had forgotten to bring salt with us; but I soon proved to my merry companions that hunters can find a good substitute in their powder-flasks. Our salt on this occasion was gunpowder, as it has been with me many a time; and to our keen appetites, the steaks thus salted were quite as savory as any of us ever found the best cooked at home. Our fingers and mouths, no doubt, bore marks of the "villanous saltpetre", or rather of the charcoal with which it was mixed, for plates and forks we had none; but this only increased our mirth. Supper over, we spread out our blankets on the log floor, extended ourselves on them with our feet towards the fire, and our arms under our heads for pillows. I need not tell you how soundly we slept.[11]

Undoubtedly, Audubon drew the long-billed curlew at this time although Havell's date of the print is 1834 which indicates the time lapse between Audubon's water color painting and the engraving. Audubon would send his watercolors and sometimes fragments by George Lehman or Maria Martin to be engraved by Robert Havell, Jr., in London.

The young Swiss painter, George Lehman, [12] who accompanied Audubon to Charleston, painted the "View of Charleston" in the background of the painting of the long-billed curlew and it is included in the original watercolor that is preserved in the New York Historical Society (Figure 4). Lehman certainly made a copy of the background for his own use. A view of Castle Pinckney (erected in the late 1700's and named for the distinguished South Carolinian, General Charles Cotesworth Pinckney) which appears in the Charleston background painting, was located in Eliza Bachman's album entitled, "The Friendly Repository and Keepsake of Miss Mary Eliza Bachman", signed: "Geo. Lehman to Miss Mary Eliza Bachman Nov^ber^ 1831". The view in the album is an exact segment of the larger view of Charleston which was done from Haddrell's Point in Mount Pleasant for the Audubon painting.

Lehman obviously kept a copy of the large background for when he left Audubon's employ and returned to Philadelphia, he made a lithograph, "Charleston South Carolina From Fort Pinckney, 1832-1833" (Figure 5). This lithograph again shows the Charleston background almost identical to the one in the background of the long-billed curlew painting. Many of the structures can be specifically identified. To my untrained eye, using the View of Charleston, South Carolina in 1850 and a map of Charleston Harbor in 1863, I would identify the following structures: the first steeple directly in back of Castle Pinckney is St. Michael's Church; the second steeple as one looks to the right is the Circular Church; the third steeple is St. Philip's Church. In the foreground, between the steeples of the Circular Church and St. Philip's, would appear to be Vander Horst Row. Under the bill of the curlew are cotton barges.

Lehman also painted other South Carolina landscape backgrounds for Audubon's paintings. In the painting of the Snowy Heron (Plate #242) the painting is subtitled, "Rice Plantation" (Fig-

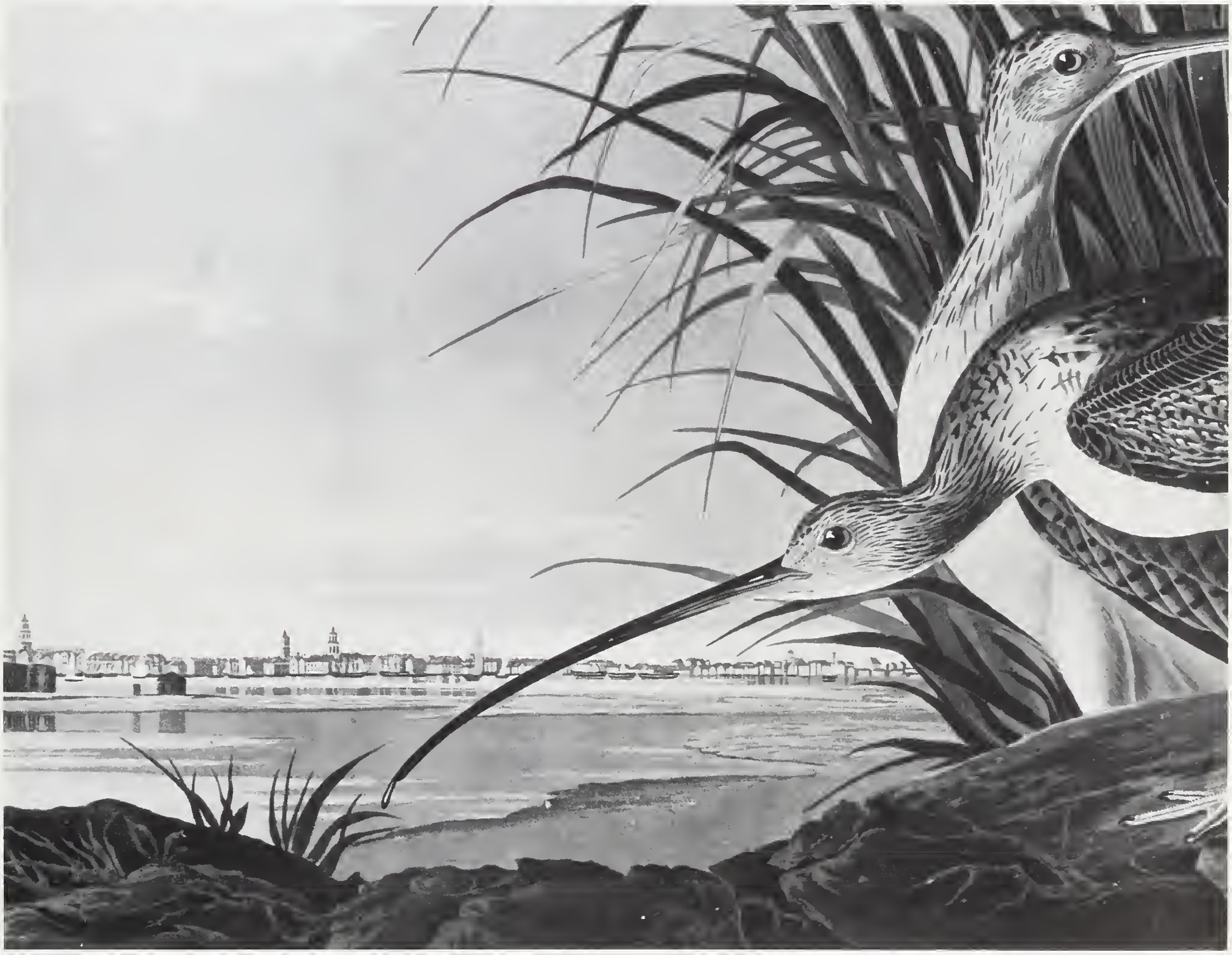

FIGURE 4. The "View of Charleston", by George Lehman, was placed in the background of Audubon's portrait of the long-billed curlew (Detail).

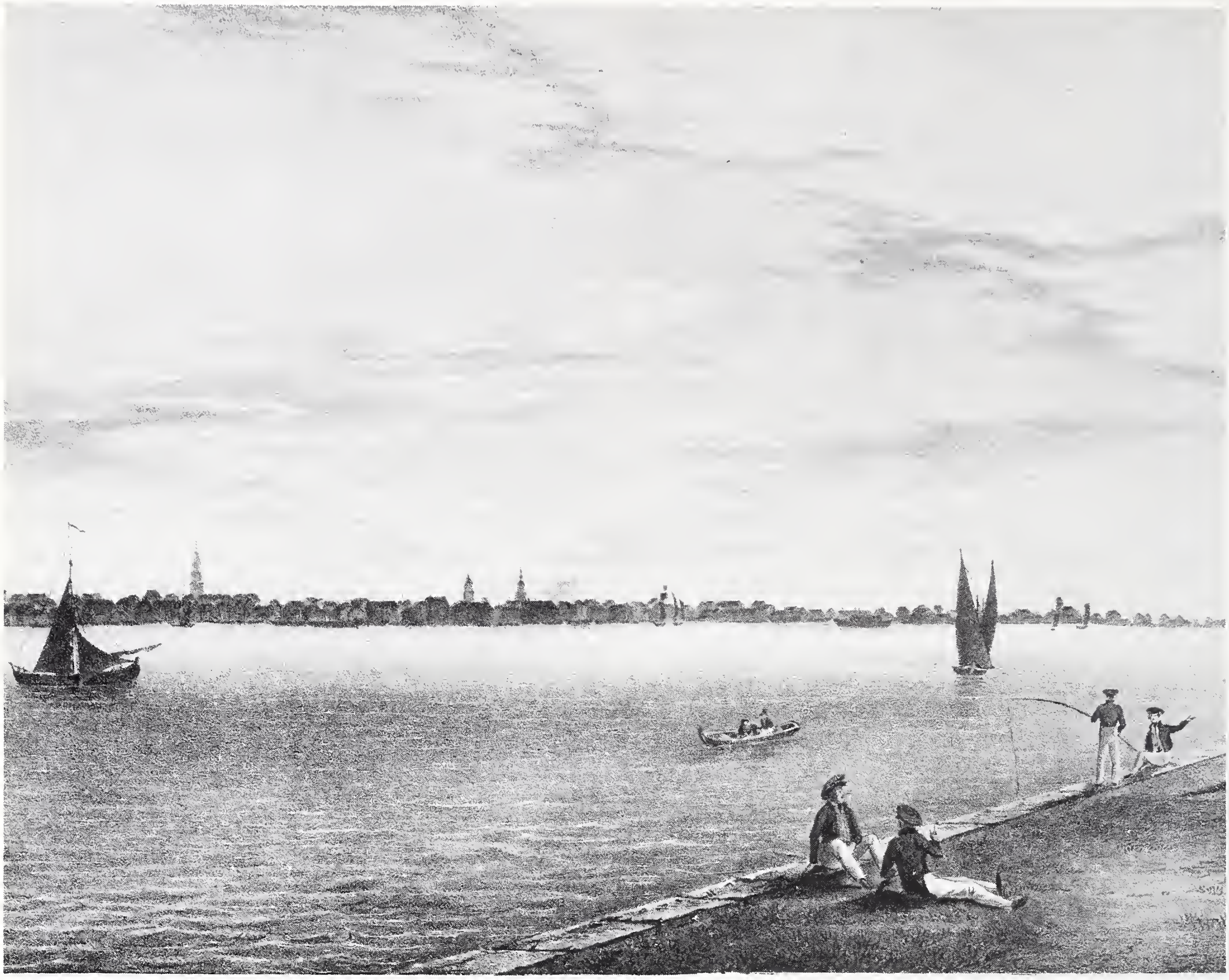

FIGURE 5. Lehman's subsequent lithograph, "Charleston South Carolina from Fort Pinckney", presents a Charleston background almost identical to the one in the background of the long-billed curlew painting.

ure 6). It has been suggested by Anna Wells Rutledge that this house is the Heyward House on James Island (built in 1719 by Thomas Heyward).[13] Also in the background is a small figure of a man with a gun; perhaps this is Audubon himself. Still another background drawn by Lehman is the one in the plate of the Common Snipe (Plate #243); the river is supposed to be the Santee River and the painting shows two houses on a South Carolina Plantation (Figure 7).

CONCLUSION

Audubon's painting of the long-billed curlew with its background of Charleston is a tangible reminder of Audubon's feeling for the City of Charleston, his relationship with the Reverend John Bachman, and his relationship with many of his physician-naturalist friends.

Since I feel that this painting embodies many of the interests of Dr. Joseph I. Waring[14, 15] — Charleston, birds, the habitat, and the role of Charleston physicians in the scientific community of the times — I would like to present this print (Figure 4) of the Waring Library Society.

ACKNOWLEDGEMENTS

Anne Donato, Anna Wells Rutledge, Edward Dwight, and Virginia Rugheimer supplied information for this article. The Wintherthur Museum provided photographs from Mary Eliza Bachman's memory book; the New York Public Library supplied duplicates of Lehman's lithograph of Charleston from Fort Pinckney; Hillel Berger photographed the Audubon plates from a copy in the Houghton Library, Harvard University; and the Fogg Museum photographed portraits of Bachman and Audubon and plates from Holbrook's *North American Herpetology* from material in the Power-Gifford Library. ☐

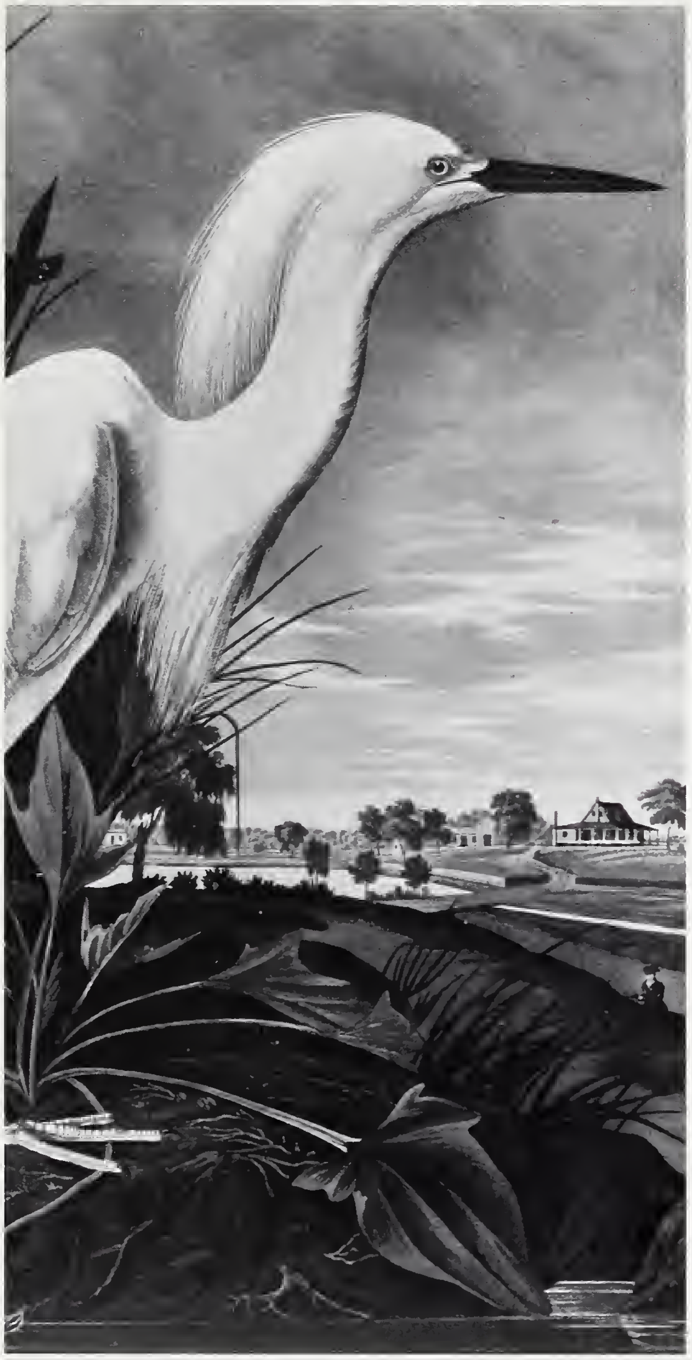

FIGURE 6. Lehman's painting of the Snowy Heron contains a background, by Lehman, of a South Carolina plantation (Detail).

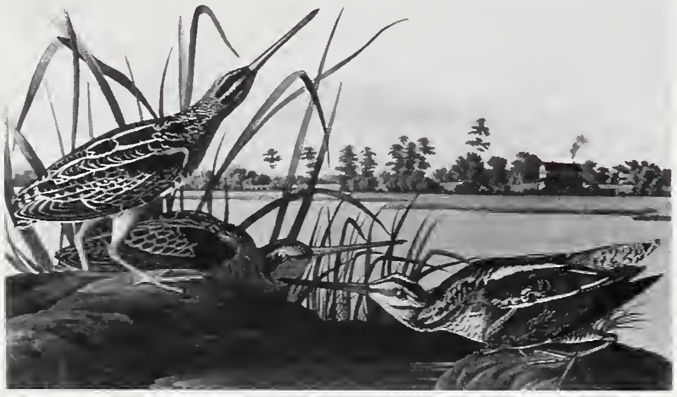

FIGURE 7. Another South Carolina plantation, possibly on the Santee River, appears in the background of the Audubon painting of the Common Snipe (Detail).

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ROBERT MILLS' SOURCES FOR THE SOUTH CAROLINA LUNATIC ASYLUM, 1822

JOHN M. BRYAN*
JULIE M. JOHNSON**

On 22 July 1822 in the morning before the heat of the day, the cornerstone was laid for the South Carolina Lunatic Asylum in Columbia. The ground-breaking was attended by a "respectable concourse of citizens," who then marched in procession to the nearby College Chapel for speeches. A dinner for dignitaries concluded the ceremonies with everyone basking in "sentiments of sociability, humanity and benevolence which the occasion was so well calculated to excite."[1]

This asylum has long been recognized as a major work by Robert Mills (1781-1852) and as an early structure designed specifically for the treatment of the insane.[2] Recent research brings into focus the sources of Mills' design and thus locates the architect and the building more firmly in the evolution of American psychiatric medicine and architecture.

The first patient was admitted to the Asylum on 12 December 1828. Prior to that time a number of hospitals had been built in America specifically for the treatment of the insane. Notable among these were the Eastern State Hospital, Williamsburg, Virginia, designed by Robert Smith and opened in 1773. Thomas Jefferson, in his famous architectural critique of Williamsburg, deemed this hospital and the College of William and Mary to be "rude, mis-shapen piles, which, but that they have roofs, would be taken for brick-kilns."[3] Another antecedent of Mills' design, the Friends' Asylum, located in Frankfort, Pennsylvania, was said to have been "patterned on the lines" of one at York, England.[4] The Philadelphia Society of Friends sponsored this building which opened in 1817. The following year the Asylum for the Insane (later called the McLean Asylum) opened in Charlestown, Massachusetts. This facility was designed by Charles Bulfinch who, in preparation for the work, had visited both New York and Philadelphia to study hospital plans. In New York the Bloomingdale Asylum was planned in 1816 and opened in 1821, having been designed by Thomas C. Taylor. Taylor's plan, like that of the Friends' Asylum in Pennsylvania, was influenced by English precedents. The same is true of The Retreat, the asylum in Hartford, Connecticut, which was begun in 1821 and opened in 1824.[5]

Excepting the Williamsburg hospital, all of these early American antecedents of Mills' South Carolina Asylum were influenced by avant garde theories of psychiatric treatment and asylum design, theories developed largely by the English Quaker William Tuke (1732-1822) and widely disseminated by his grandson Samuel Tuke (1784-1857).[6] In 1791 William Tuke visited an acquaintance in an asylum at York. The conditions there prompted him to launch a reform movement which literally unfettered psychiatry in England. Godfrey Higgins (1773-1833), an associate of the elder Tuke, has left us a vivid description of the conditions at the York asylum prior to the reform:

> I went early in the morning to examine every place; after ordering a great number of doors to be opened, I came to one which was in a retired situation in the kitchen apartments, and which was almost hid by the opening of a door in the passage; I ordered this door to be opened; the keepers hesitated, and said they had not the key; I ordered them to get the key, but it was said to be mislaid, and not to be found at the moment; upon this I grew angry, and told them that if they would not find it, I could find a key at the kitchen fire-side, namely the poker; upon that the key was immediately brought. When the door was opened, I went

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> into the passage, and I found four cells, I think of about eight feet square, in a very horrid and filthy situation, the straw appeared almost saturated with urine and the walls were daubed with excrement. The air holes in each cell, were partly filled with it. I asked the keeper, if these cells were inhabited by patients? and was told they were at night. I then desired him to take me upstairs, and shew me the place of the women, who came out of these cells that morning. He shewed me into a room the size of which he told me was twelve feet by seven feet ten inches, and in which there were thirteen women, who he told me had all come out of these cells that morning. I became very sick, and could not remain longer in the room.[7]

These conditions were the norm. William Tuke resolved to do something about it, and under his leadership the Quakers opened their own Retreat at York in 1792. Here he developed and refined notions of treatment and design. Working from the York experience, William's grandson Samuel published *Practical Hints on the Construction and Economy of Pauper Lunatic Asylums* (York: William Alexander, 1815). Here he presents the guidelines published for the Wakefield Asylum design competition as well as the winning design of William Stark (? -1813) and an essay relating his own convictions concerning treatment to Stark's Wakefield plan. In short, Samuel Tuke's *Practical Hints* couples the form and function of buildings intended for the treatment of the insane.

Architecturally speaking, asylums had been prison-like places of confinement prior to Tukes' work, but they believed that the accepted practice of solitary confinement was pernitious, for in such a situation ". . . patients will become indifferent, even to considerable privations; that sense of honour and love of liberty, which, when judiciously appealed to, have so powerful an influence in promoting self-government, will be weakened if not extinguished; and degradation of character, with a state of general insensibility, ensues, to which the common observer, and the idle and ignorant attendant, often give the appellation of comfortable."[8] At the other extreme, Samuel deplores ". . . the practice of herding large numbers of insane persons in one apartment," creating scenes of "turbulence and confusion" under the guise of furnishing "amusement to the patient."[9] Having observed such a scene he notes that ". . . in the midst of society, every one seemed in solitude . . . society could not exist in such a crowd."[10] Furthermore, from a "curative point of view" it was impossible to observe "carefully the vacillations of each patient's disease when they are visited en masse."[11] He concludes that small groups of ten to fifteen constitute the ideal day ward room. In addition to day rooms of limited size, he recommends that insofar as possible every patient be provided a private bedroom. These bedrooms and day rooms must be related to one another in such a way as to insure supervision and to facilitate the transfer of patients from one room to another. The passages must provide well lighted corridors which may serve as exercise galleries during inclement weather. Furthermore, the facility must insure segregation by sex and symptom or "degree" of disease, offer the opportunity for therapeutic work and outdoor exercise, and every effort must be made to render the building both "cheerful and calculated for voluntary change of place and scene."[12] Too often, he notes, "cheerfulness" is sacrificed to security when with proper attention to design this need not happen. For instance metal window frames, glazed with panes are suggested as a replacement for the traditional iron bars and wooden or metal shutters, and each day room is to be heated by hot water or hot air if possible and is to have access to interior privies.

The integration of architectural and therapeutic ideals advocated by William and Samuel Tuke was widely publicized during the opening years of the 19th Century, and there can be no doubt that their work was known in America. On 30 November 1815, a physician from New York, John W. Francis, wrote in the *Visitors Book* of the York Retreat that:

> J. W. Francis is not wholly ignorant of the State of the Lunatic Asylums in North America, and he has visited almost all the institutions for the insane in England. He now embraces this opportunity of stating that this establishment far surpasses anything of the kind he has elsewhere seen, and that it reflects equal credit on the wisdom and humanity of its conductors. Perhaps it is no inconsiderable honour to add that institu-

> tions of a similar nature and on the same plan are organizing in different parts of the United States. The New World cannot do better than imitate the old so far as concerns the management of those who labour under mental derangement.[13]

The Tukes' experience at York was disseminated when Samuel published his *Description of the Retreat* in 1813. The following year it was reviewed by Sydney Smith in the *Edinburgh Review*. He praised the Quakers' benevolence and said "in this instance they have set an example of courage, patience and kindness, which cannot be too highly commended, or too widely diffused, and which we are convinced will gradually bring into repute a milder and better method of treating the insane."[14] The Tukes' example was "widely diffused," for in 1815 a prominent London doctor observed that "already the original stimulus of the Retreat, and the report of a new institution for the cure of the insane, have had a most beneficial effect upon the private establishments near London. They now endeavour to make a boast of their care and attention to the poor lunatics."[15]

In America the early asylums — McLean, Frankford, Bloomingdale and Hartford — adopted many of the Tukes' suggestions. This is not surprising, for the Quaker community was both influential and international. Indeed, the Quakers in Philadelphia published an American edition of Tukes' *Description* in 1813. More to the point, in 1814 they published an *Account of the Rise and Progress of the Asylum, Proposed to be Established near Philadelphia, for the Relief of Persons Deprived of the Use of Their Reason. With an Abridged Account of the Retreat, a Similar Institution near York, England.* And in 1815 Samuel Tuke wrote "A Letter on Pauper Lunatic Asylums" for Thomas Eddy who was then trying to establish the Bloomingdale Asylum; the directors of that hospital published Tuke's letter in 1815.[16] In 1839 Pliny Earle, a prominent psychiatrist and early superintendent of Bloomingdale, wrote that "the Retreat near York has long been quoted in the United States, as approaching nearer to perfection in its management, and as giving a higher per centage of cures than any other establishment.[17]

Robert Mills was an eclectic, omnivorous reader; moreover, during the period 1815-1820 he spent a great deal of time in the Philadelphia area. He must have read Samuel Tuke's writings, for in both conception and in detail the South Carolina Lunatic Asylum reflects English prototypes — point for point there is a congruity between what Tuke recommended and what Mills designed. In the South Carolina building the metal window frames, the bedrooms, corridors, day rooms, heating system, and exercise courts all meet Tuke's criteria. But Mills modified previous ground plans to achieve his ends, and the derivation of these alterations is especially notable. The wings of the Carolina Asylum met the central block at an oblique angle. Mills envisioned that subsequent additions would result in a hemispherical, horseshoe shaped enclosure, the extended wings of the building embracing a garden. This configuration was an ingenious solution to several problems at once. The new theories of humane or "moral" treatment advocated a rural setting or "retreat" in which the patient could benefit from contact with nature. Indeed, one hallmark of the Tuke influence is the inclusion of therapeutic gardens. Mills, designing for an urban site, projected a building form that would serve to screen the enclosed gardens from the city. He went further. His plan shows that he intended the flat roofs of the wings, surrounded by high parapets, to be used as roof gardens. These appear to be the earliest roof gardens in America, and they do not derive from earlier English hospital design.

The splayed wings of the South Carolina Asylum served another purpose. Tuke had declared that visual separation of the sexes was desirable. William Stark, architect of the Glasgow and Wakefield asylums, had solved this problem in the former by separating the wings with a central stairwell and keeper's quarters, largely lit by means of skylights. However, Tuke objected to this ground plan, the famous "Panopticon," for he felt that the central keepers' facilities were too cramped and unpleasant to appeal to and retain a reputable staff.[18] By the same token he admitted that the plan at York failed to provide adequate separation of the wings. Due to the urban setting, Mills could not adopt the open-ended H plan solution employed by Stark at Wakefield. Instead, in Columbia Robert Mills adapted the Panopticon — using only two of its wings, or, read another way, he pushed back the wings of the York plan. In any event, by angling the wings he broke the line of vision between the wards without resorting to physical barriers that Tuke deemed to be psychologically damaging.

Another significant similarity between the Stark-Tuke and Mills plans is found in the tightly spiraling stairways, made of masonry and cantilevered from the walls. The principal virtue of these stairways is that they are fireproof. Secondarily, they are compact and require relatively little floor area. Robert Mills introduced this type of stair construction to America in 1822 with his Records Office in Charleston, and he used it again and again throughout his career. Did he first come upon this motif in his reading about the new developments in English asylums? We do not know. However, it is fair to say that the fireproof stairways of both Mills and Stark ultimately derive from Andrea Palladio's *Four Books of Architecture* (Book I, chapter xxviii, Venice: Bartolomeo Carampello, 1601), but the derivation of this detail is the subject of another essay. ☐

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11. *Idem.*
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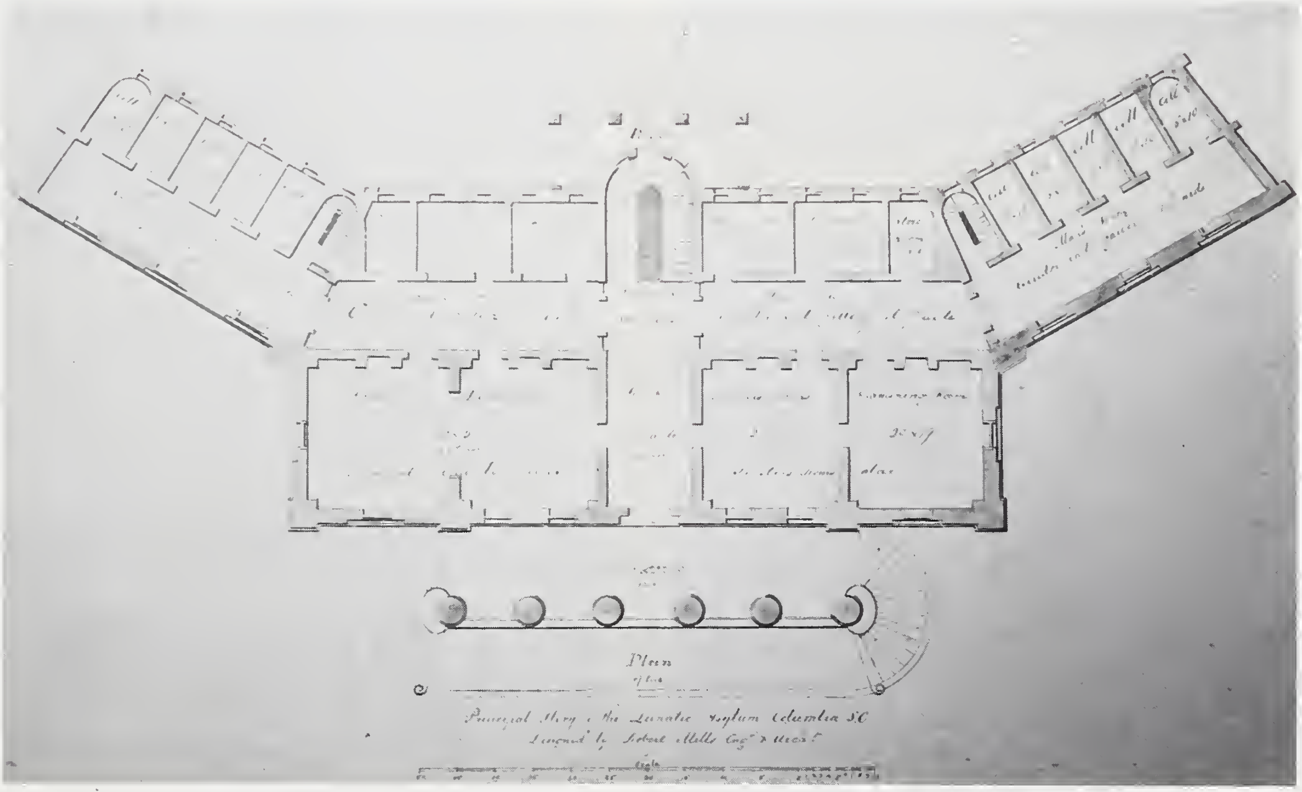
Plan
of the
Principal Story of the Lunatic Asylum Columbia S.C
Designed by Robert Mills Engr & Archt
Scale

FIGURE 1. Plan: "Principal Story of the Lunatic Asylum Columbia, S. C." By Robert Mills. From the collection of the South Carolina Department of Archives and History.

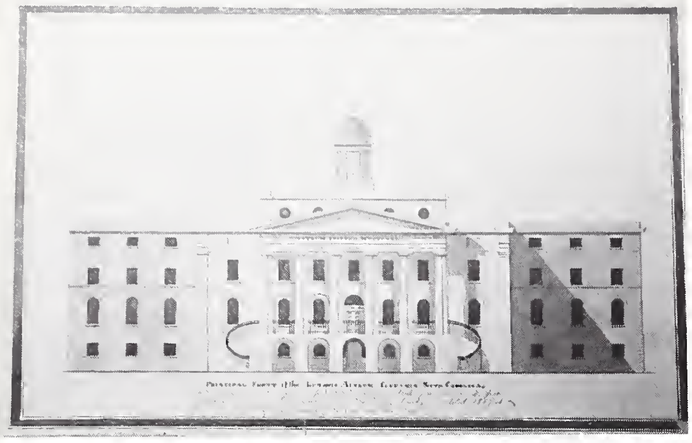

FIGURE 2. Elevation: "Principal Front of the Lunatic Asylum Columbia, South Carolina." By Robert Mills. From the collection of the South Carolina Department of Archives and History.

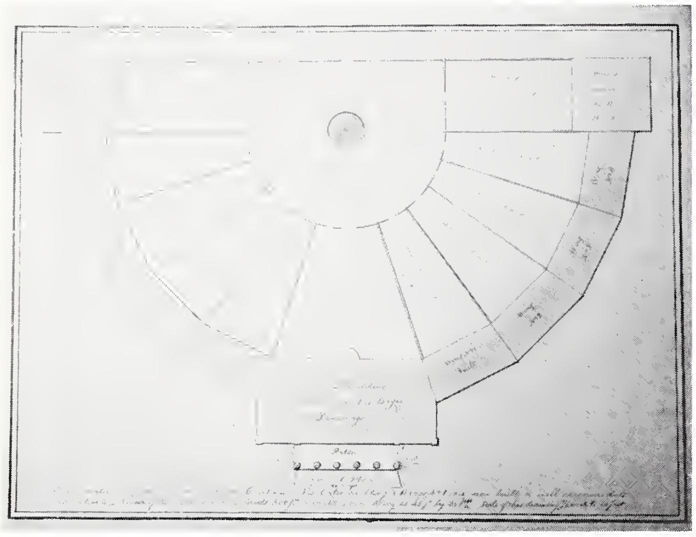

FIGURE 3. Building and Grounds Plan: "General Plan of the Lunatic Asylum Columbia South Carolina." By Robert Mills. From the collection of the South Carolina Department of Archives and History.

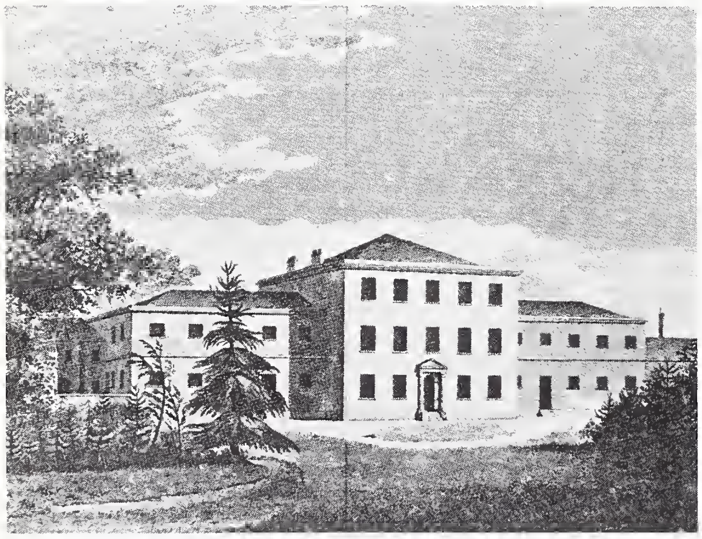

FIGURE 4. Elevation of the Retreat at York, England. From John D. Thompson and Grace Goldin, *The Hospital: A Social and Architectural History* (New Haven: Yale University Press, 1975).

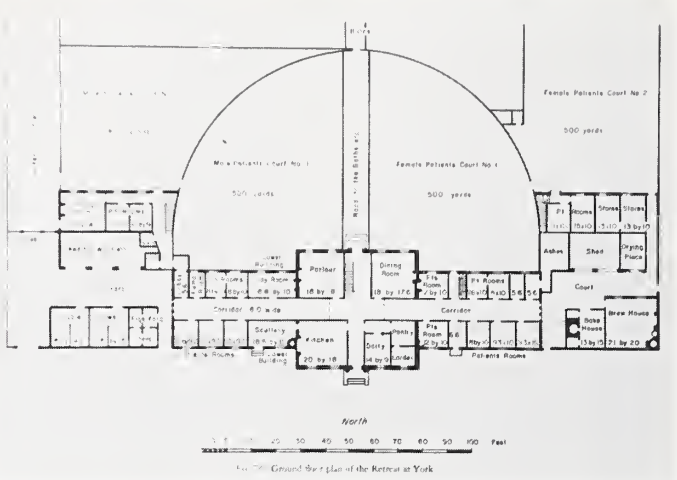

FIGURE 5. Ground Plan of the Retreat at York. From John D. Thompson and Grace Goldin, *The Hospital: A Social and Architectural History* (New Haven: Yale University Press, 1975).

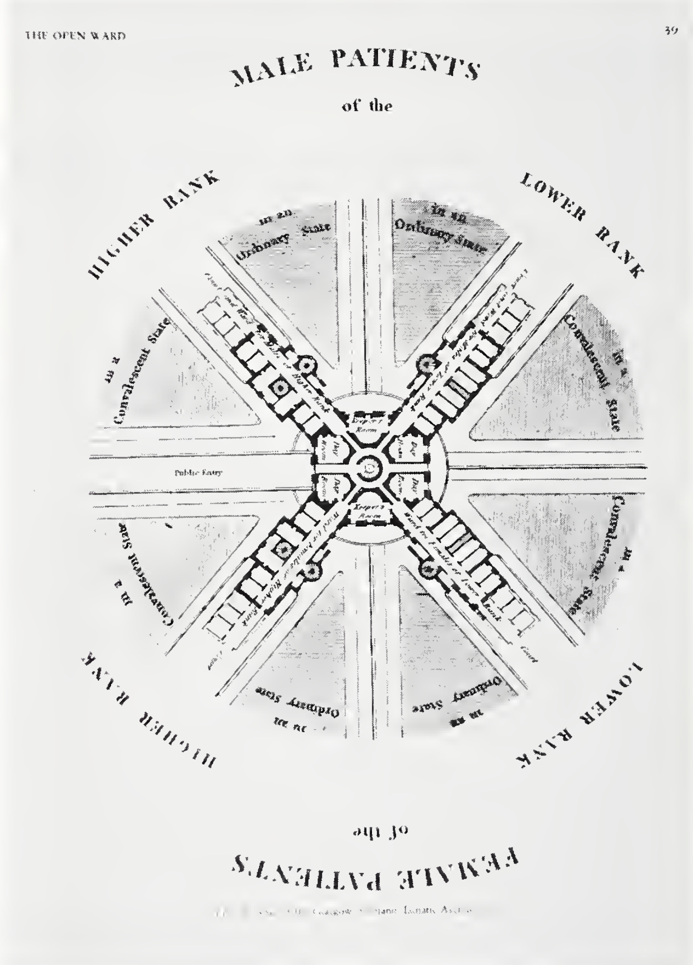

FIGURE 6. Plan of the Glasgow, Scotland, Lunatic Asylum. From John D. Thompson and Grace Goldin, *The Hospital: A Social and Architectural History* (New Haven: Yale University Press, 1975).

TUBERCULOSIS IN SOUTH CAROLINA

ALLAN D. CHARLES*

INTRODUCTION

In the 19th century tuberculosis was given little attention by South Carolinians. By accidents of mild temperature and low humidity, Aiken, South Carolina, developed after 1840 into a tuberculosis resort, but the patients came overwhelmingly from the North and usually remained at Aiken only during the winter. The town evolved into a general winter playground for wealthy Northerners by the turn of the century, and only Aiken Cottages, a small, privately-endowed tuberculosis sanatorium opened in 1897, served as a reminder of Aiken's initial claim to fame.

As the end of the 19th century approached, however, South Carolina began its first industrial spurt, and tuberculosis cases began to increase simultaneously. A direct cause and effect relationship is difficult to demonstrate, but the F.C.S. Bradbury study in 1930-31 concluded that, given open cases of tuberculosis, "the most important social factors [in the spread of the disease] were poverty, undernourishment, and overcrowding in dwellings" — all of which were conspicuously present in the early stages of industrialization. South Carolina's tuberculosis death rate per 100,000 population climbed from 97 in 1870 to 196 in 1890. The rate declined spontaneously to 166 in 1900 and to 121 in 1920. A concerted social attack on the disease began in the second decade of the 20th century and undoubtedly hastened the attenuation of tuberculosis to 78 per 100,000 in 1930 and 48 in 1940. With the advent of drug therapy in the 1940's the rate per 100,000 dropped to 16 in 1950 and to less than 6 in 1960.[1]

As medical historian George Rosen has pointed out, the great reduction in tuberculosis in the United States may be partially ascribed to general improvement in living standards, nutrition, housing, and cleanliness, but the role of governmental and social agencies concerned primarily with attacking the disease cannot be discounted. In South Carolina these agencies had had a definite impact on tuberculosis (especially among the white population) by 1940. The story of how people in South Carolina came directly to grips with the ancient scourge is interesting in itself, but what is especially intriguing about the South Carolina saga is the utter disregard for any hard lines of separation between public and private efforts.

In South Carolina, for example, the State Board of Health (established by the General Assembly in 1879) was identical with the South Carolina Medical Association, a private body. The board was run by an executive committee nominated by the SCMA and appointed by the governor, an arrangement which continued with only slight modification until 1973. Thus from the very outset of modern public health efforts, there was an organic connection between the public and the private sectors. One may conclude that such liaison was not very common, as Dr. H. A. Pattison of the National Tuberculosis Association remarked in 1925 that the South Carolina arrangement was "singular."[2]

BOVINE TUBERCULOSIS

After Robert Koch in Germany isolated the tubercle bacillus in 1882, there was new interest in a possible conquest of tuberculosis. While no miracle cure was forthcoming, the work of Koch and Pasteur had made it obvious that tuberculosis could be transmitted to humans through the milk of infected cows. It was on that front that a South Carolinian first struck against the disease.

Dr. Leon Banov, first as health officer for the city of Charleston and later as head of the city and county combined health office, noted in the 1890's that one death in six in his city was due to tuberculosis. To eliminate milk as a source of infection for various diseases, including tuberculosis, he successfully campaigned to impose

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cleanliness on the dairies supplying milk to the city. By 1915 he was able to hold milk producers to the requirement that all milk sent to the city had to come from tuberculin-tested cows.[3] Not until 1925 was Chicago's Cook County placed under similar requirement.

Dr. T. G. Simons, chairman of the executive committee of the State Board of Health at the turn of the century, was not far behind the redoubtable Charlestonian. In 1899 he proposed that the legislature pass an act calling for testing and isolating tuberculous cows and destroying them when possible. The General Assembly was slow to act — primarily because the poverty of the state forbade reimbursement to farmers for the destruction of valuable livestock. By 1914 the Sanitary Code of South Carolina did contain a law requiring that all dairy "cows . . . should be tested for tuberculosis once a year." Not until the period of World War II, however, were tough laws really enforced on bovine tuberculosis.

PULMONARY TUBERCULOSIS: RESISTANCE COALESCES

While the definitive Park-Krumwiede report of 1912 proved that much scrofula, intestinal tuberculosis, and tuberculosis in children was due to contaminated milk, the fact remained that pulmonary tuberculosis in young adults was exacting the greatest social cost. The disease was endemic and ubiquitous, and in the early 20th century science offered little help. The technique of pneumothorax was described before the South Carolina Medical Society at Charleston in 1911, and section of the phrenic nerve was practiced in the state by the early 1930's, but the "rest cure" was still the main line of defense against the "great white plague." Thus the founding of a state sanatorium was the nucleus around which the resistance effort coalesced.

The catalyst for the foundation of a TB sanatorium was the increasing incidence of tuberculosis among the inmates of the State Hospital for the Insane and the State Penitentiary in the 1890's. Here were two consolidated groups of people, wards of the state and therefore under observation, with tuberculosis rampant among them.

Dr. James W. Babcock, superintendent of the State Hospital, continually complained in his annual reports to the legislature that his facilities were overcrowded and that there was no special ward in which to separate tuberculosis sufferers from the other patients. In the State Hospital 298 patients died of tuberculosis between 1880 and 1893. Tuberculosis mortality peaked in 1900 when 105 out of the 230 deaths at the asylum were caused by the disease. In the State Penitentiary 65% of all deaths were due to tuberculosis during the period 1897 through 1899.[4]

Dr. A. A. Moore of Camden, a member of the Board of Trustees of the State Penal and Charitable Institutions, may have been the first to suggest the obvious. In a report to the legislature in 1899 he advocated construction of a separate sanatorium for tuberculous mental patients. The idea grew in his mind, and in 1903 he read a paper at the South Carolina Medical Association's annual meeting in which he noted that Massachusetts had pioneered with the first state tuberculosis hospital in 1898. South Carolina should follow suit.

Under the impetus of two successive legislative investigations of conditions (including crowding) at the State Hospital, construction for the institution's expansion was begun by 1913 on a large tract of land some six miles northeast of Columbia. For several years there was some confusion in the minds of Babcock and his successor as to whether the General Assembly meant simply to construct a tuberculosis facility or to move the entire State Hospital to the country and abandon Bull Street.

The first construction northeast of Columbia evolved into the modern Crafts-Farrow facility of the State Hospital after serving many years as the Palmetto Division for black patients of the hospital.[5] Tuberculosis was cut out of this initial expansion, and there was some doubt as to whether Governor Cole Blease, who had a reputation as a non-spender, would sign a bill requiring that more money be expended to construct a separate, state tuberculosis facility. Dr. Robert Wilson, chairman of the Board of Health and a leader in the sanatorium movement, heartily recommended such an institution, adding that "perhaps the very magnitude of the problem makes any effort appear almost hopeless."

Meanwhile, a longtime proponent of the sanatorium idea, Dr. John L. Dawson, president of the South Carolina Medical Association, professor at the South Carolina Medical College, and himself a tuberculosis sufferer, was working to get a sanatorium bill through the legislature.

He found a ready ally in a fellow tuberculosis victim, Richland County representative George R. Rembert. Rembert died before his bill reached the governor, but his widow, Mrs. Annie I. Rembert, personally set the legislative act on Blease's desk. It had been rumored that the governor would veto it, but the personal appeal had its effect. South Carolina was to have a state sanatorium.[6]

The first year's appropriation was $11,000. On a tract of land two miles northeast of Crafts-Farrow the first buildings constructed were a frame structure for white, male patients and a frame residence for the superintendent. The South Carolina State Sanatorium opened in June, 1915. In 1920, when a post office was installed, the name "State Park" was hit upon, and this designation eventually superceded the official one. The sanatorium's first patient was Arthur Jones of Ridge Spring. He was reported to have become so lonesome among the pine-clad sand hills that he left the institution twice before other patients arrived to keep him company.

Mr. Jones and his growing number of fellow patients were looked after by a staff of six headed by Dr. Ernest Cooper, who served as superintendent from the inception of the sanatorium until he died in office in 1939. A native of Charlotte, North Carolina, Cooper received his M.D. degree from Johns Hopkins in 1910. This staunch Presbyterian, kindly but resolute, was to leave his mark on the evolution of State Park. He was a stout defender of his turf and got into trouble in 1917 for his practice of shooting the dogs of trespassing hunters.

While the state was overcoming its inertia in deciding to build a sanatorium, one of its counties had actually carried off the laurels for being the first to establish a publicly-supported tuberculosis facility in South Carolina. The movement was, however, seeded with private funds.

Across the street from the State Capitol, the Daughters of the Holy Cross of Trinity Episcopal Church set aside $500 in 1913 for a tuberculosis camp. Rising to the challenge, the Richland County delegation to the General Assembly matched the sum with $1000 in the county supply bill, and in 1914 Ridgewood Sanatorium came into existence. Ridgewood began as a tent encampment (perfectly all right in the view of the fresh air therapists of the day) but later grew into a village of tiny cottages on donated land outside of Columbia. Many individual cabins were constructed by various charitable organizations, and the sanatorium was the chief project of the Richland County Tuberculosis Association, which actually operated the institution.

That the sanatorium movement was widespread in the mid teens was shown by the development of the Hopewell Sanatorium in Greenville. There a local civic activist, Mrs. M. P. Gridley, first president of the Greenville Tuberculosis Association, was credited with being the founder of Hopewell in 1915, the same year that the State Sanatorium opened. Hopewell was receiving city and county funds by 1925 and in 1930 merged with the newer Greenville County Tuberculosis Sanatorium.

1915 marked a turning point in South Carolina's approach to pulmonary tuberculosis. From 1899 on, the Board of Health had periodically issued bulletins to acquaint people with the symptoms of the disease, but it was admitted at the time that few were probably reached by the bulletins or by the Board's booth at the State Fair. There was, moreover, no place to isolate and treat such victims as were found, and there was no concerted program to eradicate the disease. Only bovine tuberculosis had been under consistent attack prior to 1915. The foundation of three sanatoria by 1915 provided the necessary nuclei for an accretion of forces against "consumption."

Dr. James A. Hayne, state health officer, outlined to the legislature in 1916 the plan of attack which came to be generally followed: (1) Establish free clinics in the larger cities for diagnosing cases. (2) Require physicians to report all cases to the health authorities. (3) Employ district nurses to visit sufferers and tell them how to protect themselves and others. (4) Sponsor county tuberculosis camps to isolate advanced cases. (5) Create a large, state sanatorium for incipient and curable cases.

PULMONARY TUBERCULOSIS: THE BATTLE JOINED

The great volunteer allies of the State Board of Health were the anti-tuberculosis associations. In 1904 the "National Association for the Study and Prevention of Tuberculosis" was formed, but that ponderous nomenclature was changed in 1918 to the "National Tuberculosis Association." Some $3000 of seed money from the national organization helped the South Carolina Tuberculosis Association to get started in 1917.

At its 1908 meeting the South Carolina Medical Association laid plans for the organization of anti-tuberculosis associations in the counties. By 1918 there were four such organizations, and by 1940 all but seven counties had tuberculosis associations or committees.[7]

The state level South Carolina Tuberculosis Association evolved from the Red Cross Christmas Seal Commission in 1917 when enough funds had been accumulated to warrant year round anti-tuberculosis work by a separate organization. The Christmas seals became the main source of financing for the new SCTBA. Dr. L. A. Riser, head of the Board of Health's Rural Sanitation Department, served as executive secretary the first year and was succeeded by Miss Chauncey Blackburn (soon Mrs. D. McDonald) who headed the organization from 1918 until it virtually worked itself out of a job in the 1940's. Mrs. McDonald also served as field secretary for State Park for a few years in the early 1920's.

J. Nelson Frierson, Dean of the University of South Carolina Law School, oversaw the SCTBA and boosted Christmas Seal sales as president of the organization during its period of great activity, 1920-1942. The long-lived team of McDonald and Frierson meant that there was great continuity at the top.

The inter-relationship of the public and private sectors was apparent in the "interlocking directorate" of the SCTBA's officers. Honorary vice-presidents included Dr. James A. Hayne, state health officer; Dr. Robert Wilson, president of the Board of Health; and Dr. Ernest Cooper, superintendent of the State Sanatorium.

One of the first on-going projects of the SCTBA was to assist the Board of Health in its fledgling effort at providing public health nurses. Mrs. Ruth Dodd and Miss Helen Fenton were pioneers supported by both organizations.[8]

The SCTBA began its program of following up known cases of tuberculosis by working with a set of some 900 young men who might not have known they were tuberculous but for a new government practice — the army induction physical. These World War I draft rejects composed a ready-made set of referrals for the young association.

Knowledge that tuberculosis fell especially heavily on the black population was officially recognized as early as 1903. While the obvious environmental problems of blacks — crowding, poor hygiene, etc. — must have contributed to their disproportionate share of tuberculosis cases, a modern tuberculosis specialist, Dr. J. W. Fouché, director of the State Park Health Center, recently maintained that the genetic factor cannot be ruled out. His experience in New York taught him that Jews are particularly resistant to the disease, apparently due to their lengthy history of contact with the bacillus in the Middle East and the Mediterranean. Blacks, by contrast, had comparatively little contact with the scourge in sub-Saharan Africa and have yet to evolve a substantial immunity.

Plans for a black "Palmetto Sanatorium" went ahead to completion in 1920. Sometimes called the "Palmetto Division," it was occasionally confused with the State Hospital's Palmetto Division (later Crafts-Farrow) for blacks some two miles away. The Palmetto Sanatorium was never actually administered separately, being under the direct supervision of Cooper and his staff.

Mrs. Annie Rembert served until 1920 as State Park's first field secretary. She was convinced of the need to work with both races in fighting tuberculosis and assisted the movement to establish the Palmetto Sanatorium. Her field work included interviews or correspondence with all editors in the state, correspondence with prominent tuberculosis workers in every county, letters sent to all South Carolina mayors, and contacts with a long list of legislators. In 1918 Mrs. Rebecca H. Walton, a black, was hired by the sanatorium to carry on field work among the Negroes.

Meanwhile the SCTBA in 1918 began its own work among the blacks. A black auxiliary organization was set up, the "Palmetto Association," and Louise Holmes of Florence, a black, was employed to do the educational work. During her first year on the job she made 48 health talks to 8928 people, enrolled 7311 children in "health clubs," received contributions, and distributed literature.

This well organized field work hurried many tuberculous individuals of both races into the sanatoria, but the fifth point of State Health Officer Hayne's program — that State Park should be a facility devoted to incipient and curable cases — was never implemented. As early as 1916, nineteen "far advanced" cases were admitted to the State Sanatorium, and an inability or unwillingness to segregate early from advanced cases in separate institutions characterized the sanatoria throughout their history. This situation stemmed from an ambivalent attitude as to the proper function of a sanatorium. Was it a place to cure people, or was it an isolation camp for the incurable? Before the advent of drug therapy, after which most cases could be contained if not actually cured, the question was never answered. The maintenance of separate wards for advanced cases so as to preserve higher morale among the curable was the only compromise.[9]

State Park, very early in its history, filled up the free beds furnished by state funds. To support more beds and to by-pass the cumbersome and often unrewarding process of seeking ever larger appropriations from the legislature, the sanatorium pressed individual counties, cities, and industries for funds for specific beds. Colleton County was the first county to fund a bed, and accordingly a bed was set aside specifically for a patient from that county. Similarly Columbia was the first city to co-operate, and Pacific Cotton Mills in that city was the first industry to finance a bed for its employees. Voluntary contributions by public and private organizations increased with the years until the state ultimately took on more of the responsibility itself.

By 1920 space and beds were both heavily occupied at State Park, and the field secretary (who also handled admissions) was placing "urgent cases" in county facilities. The State Hospital had its own tuberculosis pavillion after 1918 and did not greatly tax the State Park resource. That fact was rather ironic, considering that the original impetus for building State Park had come from Bull Street.

Crowded conditions at State Park stimulated the building of local sanatoria. Camp Alice in Sumter County was founded in the early 1920's through joint efforts of a Red Cross TB committee and the city of Sumter. In Richland County Ridgewood Camp expanded to accommodate most of the county's sufferers, in fact accepting all Richland's cases in the years 1920, 1921, and 1923.

Charleston Countians were also irritated by the waiting list for State Park. During this formative period of the anti-tuberculosis movement in the late teens and early 1920's, Charleston was conspicuous by its relative absence from participation in in-patient tuberculosis work. Dr. William A. Smith of that city chided "the state's medical center" for its comparative inactivity, and Dr. H. A. Pattison of the National Tuberculosis Association noted in 1925 that the Medical College's teaching of tuberculosis was "very limited" (and that in a state which ranked ninth in the nation in tuberculosis mortality at the time).

Charleston's formidable public health officer, Leon Banov, soon moved the port city out of the doldrums. Procuring city-owned property some six miles north of the city limits, he took personal charge of two carloads of government surplus building materials from Fort Jackson and, inspiring new life into the lethargic local tuberculosis association, constructed Pinehaven Sanatorium in 1924. The county's responsibility was limited to furnishing the land and a sum of $40,000 for buildings. The Charleston County Tuberculosis Association actually ran the sanatorium and paid Dr. William A. Smith, the medical director, and his staff. Not until 1954, when the Charleston County Council took it over, did the powerful Charleston Association relinquish control of Pinehaven. Over the years the vast majority of tuberculous Charlestonians were accommodated in their home county, and few were sent to State Park.

By 1925 there were 369 sanatorium beds in the state, 268 for whites and 101 for blacks. Of the total, 136 were at State Park, and 70 were at the State Hospital. Pinehaven, the newest and largest local facility, boasted 60 beds, and Camp Alice, near Sumter, was the smallest with 20 beds. Proprietary sanatoria had vanished with the 19th century, but Aiken Cottages, sponsored by Northern, Trudeau-inspired, philanthropy, survived from an earlier era. It contained 18 beds occupied only in the wintertime.

By 1930 the Marion County Tuberculosis Camp at Mullins had joined the ranks with 13 beds. By then the state total had swelled to 505 beds, 274 at State Park and 231 elsewhere. Even so, South Carolina had a TB bed for every 3230 people, while the national average was one for every 1880.

The poor, in general, were well served, as the percentage of free care days (no charge being made to the patient) at the local sanatoria ranged from a low of 70% at Ridgewood to a high of 100% at Marion. Over the four year period 1927 through 1930 some 26.5% of the patient-care days were allocated to blacks, and virtually all of those days were free care.

The Duke Endowment assisted the half dozen local sanatoria in South Carolina as well as the greater number located in North Carolina. The Endowment reported in 1930 that South Carolina may have been inferior in beds per capita, but that South Carolinians were performing in the late 1920's a much larger volume of outpatient work than was being done in the sister state. South Carolina was more active in the prevention and control of the disease, particularly among blacks, and was superior in following up cases through clinics and visiting nurses.[10]

One of the largest problems faced by the public and private tuberculosis workers in the 1920's was in finding cases. Workers were able to keep the sanatoria full, of course, but their outpatient work and efforts to contain the disease were hampered by the frequent failure of physicians to report cases which they encountered. Tuberculosis was considered a "non-quarantinable" disease, but the Sanitary Code required that a "warning placard" be placed on a sufferer's house nonetheless. A tuberculous person was not quarantined on his premises, but he was prohibited from attending places of public assembly. Such social prescription perhaps accounted for some people hiding the disease "as they would a crime," according to the SCTBA, and they may even have begged their doctors to keep their secret.

As the decade of the 1930's progressed, however, Dr. Cooper at State Park became increasingly pleased with the general practitioners of the state. He noted in 1931 that they were increasing their calls on tuberculosis specialists in making diagnoses, and he found it "gratifying" in 1933 that the medical profession had made "greater demands on the Sanatorium than heretofore." Outpatients were up 20%. In 1935 he happily reported even greater patronage by GP's of the outpatient clinic.

Most outpatient work in the state was done, of course, not at State Park itself but through local clinics. Charleston had held a clinic since 1908 and was joined by Richland and Greenville counties in 1918. In the early 1920's such clinics came to be more widely and regularly held, and by 1924 the method was employed in 24 counties.

Charleston's clinic in 1924 was held irregularly, and local physicians rendered the medical treatment. At Anderson, by contrast, a clinic was held every Tuesday from four to six under the supervision of Dr. G. S. Clinkscales, a graduate of the special tuberculosis course at the Trudeau School in Saranac, New York. In 1924 Greenville also held a weekly clinic, but at Spartanburg a recently started clinic had been discontinued due to "irregular medical service." In Columbia the Richland Anti-Tuberculosis Association maintained a short, twice weekly clinic in the County General Hospital on Harden Street. Dr. J. H. Gibbs, editor of the *Journal of the South Carolina Medical Association*, headed a staff of local physicians who donated their services. Many counties, however, still had to wait their turn for chest specialists to be sent out by the Board of Health or the SCTBA.

The health crusade (education) division of the SCTBA did much work in a campaign against expectoration, a means of spreading tuberculosis bacilli. The widespread use of chewing tobacco by men and by some women meant that the problem was real indeed. Mrs. Chauncey McDonald, executive secretary of the SCTBA, despaired that "it takes something as deep as a genuine religious conversion, a soul-stirring love affair, or a doctor's decree to separate a man from his chew." The Board of Health had promulgated an anti-spitting ordinance for all public places as early as 1915, but little attention seems to have been paid to it. The sovereign remedy was already on the scene, however, and the problem of expectoration was solved by the era of World War II, by which time most people had shifted to cigarette smoking. Ironically the corrective for a means of spreading one respiratory disease came to be an agent for causing another.

PULMONARY TUBERCULOSIS: NEW WEAPONS IN THE ATTACK

The decade of the 1930's saw the emergence of modern hospital-type treatment for tuberculosis victims. Although drugs did not become availabe until the late 1940's, the thirties saw improved diagnosis and surgery become more important in tuberculosis therapy. At State Park even dental care was stressed after 1926, as absessed teeth were a means of tubercular infection. Thus a dentist became a permanent member of the staff.

Although tuberculin-testing, discovered by Robert Koch in the 1880's, had long been a medical commonplace, it was not until the 1930's that the X-ray, another valuable diagnostic tool, was improved enough and inexpensive enough to be of general use. The invention of cheap paper films in 1931 and miniaturized films in 1936 greatly enhanced the X-ray's usefulness.

State Park superintendent Cooper had called for an "X-ray outfit" as early as 1921, a sign of his progressive thinking, but such equipment was not installed until 1927. The one received that year, however, was modern and had a fluoroscope and stereoscope. Unfortunatley, no one on the staff knew how to operate it, so the machine gathered dust for about a year. Most of the local sanatoria depended on nearby hospitals for X-rays.

By 1936 the SCTBA was calling for more immediate X-ray follow-up of people with positive tuberculin tests, and the following year the Board of Health responded by fielding a mobile X-ray unit. The X-ray truck was mainly employed in those counties which did not have local health departments. Despite severe shortages of gasoline and tires the truck kept rolling all through World War II, and before the decade of the 1940's was over, three large vans were covering the state. The mass use of chest X-rays continued into the early 1960's, when the declining incidence of tuberculosis made such a technique unnecessary. X-ray screening of "high yield" groups then became the norm.

Modern techniques required modern facilities, and at State Park much of the expansion of the plant in the late 1920's was provided by private funds from women's clubs, the League of Women Voters, the Legislative Council of Women's Organizations, some men's clubs, and the Masons. Overcrowding, however, remained a problem at State Park, especially for blacks, and to make room for new admissions, Cooper promulgated a rule in 1932 that patients could remain at the sanatorium only for 18 months.

The first federal aid for State Park came with the Depression and the New Deal. A community building containing a chapel, library, exchange, barber shop, and post office was constructed in 1933 with funds from the Richland County Relief Association through the Reconstruction Finance Corporation. In 1936 federal, state, and private money combined in a program to erect new fireproof buildings to replace the old wooden ones. A $50,000 state bond issue was made with a pledge by the Masons to carry the first year's interest on the bonds. The money was supplemented with a U. S. Works Progress Administration grant of 30%.

In 1938 a truly modern hospital building, a six story, brick structure, was completed. The fireproof building, which was the culmination of Cooper's dream for a hospital-type sanatorium, was made possible by a U. S. Public Works Administration grant of a quarter million dollars matched by a third of a million in state funds.

The number of black beds then rose to 132 out of 563, but the black waiting list remained fairly long until the completion in 1951 of a half million dollar concrete and steel building for Negroes. Construction of this modern facility was assisted by federal matching funds under the Hill-Burton Act.

While the Depression era had little effect on the building program at State Park, other aspects of the anti-tuberculosis effort were less fortunate. In the 1920's the number of counties with health departments (including physicians and nurses) had been gradually increasing, despite the cessation of federal aid to certain, non-TB, health projects. The figure rose steadily to 23 in 1929, but there the number stood through the depth of the Depression. By 1935 federal Social Security funds were available for strengthening the public health programs, and the number of county health departments was soon on the increase again.

In the 1930's tuberculosis field workers employed an interesting method to arouse white support for fighting the disease among blacks. The SCTBA mentioned in 1936 the high incidence of tuberculosis among blacks and noted that, as the main source of servant labor, they were in constant and close contact with white children. Black tuberculosis was identified as a contant menace to any successful program of tuberculosis control. Three years later several county TB associations were reporting much popular interest in X-raying domestic servants, then common even in middle class white households.

In the 1930's two more local sanatoria were established. A joint operation was set up by Florence and Darlington which by 1947 boasted 101 beds. Spartanburg moved its tuberculosis sufferers out of a special ward in the general hospital and into a 65 bed sanatorium north of the city.

By 1947, however, the facilities in Aiken, Sumter, and Marion counties had closed. In the 1940's state aid was not forthcoming to facilities which were not fireproof and, while Aiken Cottages may have closed for lack of patients, Sumter and Marion were more likely terminated by lack of funds and a greater dependence on State Park. They were the first casualties among a dying breed, for tuberculosis, among whites at least, was being contained. By 1977 even State Park itself had become an out-patient facility.

As early as 1936, SCTBA president Frierson cautioned against overconfidence, noting that the disease was still prevalent among blacks. By 1939 the battle among whites was viewed as substantially won, and Dr. Martin Woodward, assistant state registrar of vital statistics, doubted if any other state had such a low white tuberculosis case rate (20.9/100,000). He added, however, that "we must do a great deal for our colored population" which was then running a case rate of 77.4 per 100,000.

Cooper died in 1939, the year of Woodward's optimistic report, and was succeeded by Col. William H. Moncrief, M.D., who had had wide experience in the army with tuberculosis treatment and hospital administration. In a speech before the Columbia Rotary Club in early 1941, Moncrief declared that South Carolina's death rate (white and black) of 43.7 per 100,000 was the lowest of any southeastern state and compared very favorably nationally.

The very significant fact was that these substantial gains had been made, and the specter of the "great white plague" beaten to its knees, before the advent of a single anti-tuberculosis drug. The accomplishment was all the more considerable for having been achieved in the period prior to World War II. It was not until after the war that living standards for either race significantly improved in South Carolina. Further, the battle was both officered and soldiered mostly by South Carolinians. There was some money and advice from the National Tuberculosis Association, to be sure, and the federal government assisted State Park's building program after 1933, but there was no federal administrative leadership in the anti-tuberculosis struggle until 1944. Assistance via the Duke Endowment cannot be considered truly extraneous, as it is a homegrown Carolina enterprise for both states.

The first drug which was effective against tuberculosis was the anti-biotic streptomycin. It was first used experimentally in South Carolina at Pinehaven in 1947 and at State Park the following year. Later INH, PAS, and other drugs were discovered, and combinations of them came to be used to combat resistant bacilli.

Work among the blacks was greatly increased in the 1950's, until even among this vulnerable segment of the population the disease was beaten back. Tuberculosis, unlike smallpox, still survives and still kills, but for a disease with no single "magic bullet" cure, it has been definitely thrown onto the defensive. Thus, today the National Tuberculosis Association survives as the Lung Association which, with its state and local affiliates, is concerned with all respiratory problems, even hay fever and air pollution. Tuberculosis alone can no longer occupy the energies of a movement which was born to fight it. □

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(Further information available from the author on request.)

President's Page

BETTER HEALTH THROUGH HEALTH EDUCATION

Those not familiar with an accurate analysis of the etiology of man's illnesses often assume that the physicians and their system are at fault when they see statistics with regard to infant mortality, longevity of life, etc. The United States compares unfavorably with small European countries and South Carolina's statistics compare unfavorably with those of the United States. Physicians recognize, however, that the health status of our people involves many factors. It involves genetics, environment, socioeconomics, individual motivation and education, *as well as medical care*. Most of these factors are beyond the control of the physician. Health care and medical care are not synonymous. We of the profession should, as physicians, promote quality medical care and, as good citizens knowledgeable in matters of health, promote projects that will improve the health status of the people of our state and nation.

In my Inaugural Address at Myrtle Beach on April 29, I spoke of the influence of *lifestyle* on disease and ill health. Modification of *harmful lifestyles* can effectively improve health status. To bring about this modification, one on one by physicians during the course of therapy, is slow, tedious and expensive. If we are to really address the issue and bring about long-term improvement, we must teach *proper lifestyles* and teach about the other factors that influence health status early in life. This can be done best by our public and private school systems.

We believe that a comprehensive health education program in elementary and secondary schools would be a tremendous benefit to all involved. For this reason, I am proposing the formation of a Task Force, composed of the Department of Health and Environmental Control, the Department of Education, hospitals, nursing homes, health professionals, consumer groups, and other public and private groups, to research the effectiveness of health education programs and make recommendations on program guidelines and development. After this study is made, the Task Force should make specific recommendations to our Legislature and to the public so that deficits found may be corrected.

I request that you — the Auxiliary, you — the physician, research the health education status in your own local school system. Ask questions of your faculty and students. Are you satisfied with the health education your child is receiving?

As for me, I think that it is time to add health to the three R's.

COMMITTEE ASSIGNMENTS

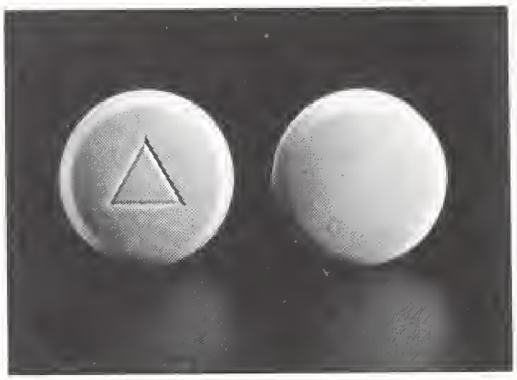
It has been most gratifying to receive so many expressions of willingness and desires to serve on the various committees. Requests for assignment on some committees have far exceeded the openings. If, by chance, you do not receive the assignment according to your desire, please be assured of my appreciation of your response and that your name will be kept on file for later appointment as vacancies occur. The new Constitution and By-Laws establishes a Commission/Committee system which should transfer most of the in-depth study of issues and problems to the various committees, thus relieving some of the work that Council has been doing and giving the decision-making process more input from our membership at large.

Your involvement is always needed. We want to hear from you.

Harrison L. Peeples, M.D.
President

The Maker

Examining a Few Myths About Prescribing.



Increasing pressure is being put on the practicing physician to prescribe drugs generically. You are told that brand–name products are universally "expensive" and generic versions are relatively "cheap." To make this case, the most extreme (rather than typical) price differentials are cited. Thus, consumers are led to believe that such differentials are commonplace. Even your knowledge and your motives as a physician are questioned.

Understandably, these views have created myths. We think it's time to examine them in the light of all the facts and ramifications.

MYTH: There are no differences in quality and performance between brand-name products and their generic counterparts. The corollary is that there are no differences among products made by high-technology, quality-conscious, research-based companies and those made by commodity-type suppliers.

FACT: The Food and Drug Administration does a good job in monitoring a generally excellent drug supply. Still, it has nowhere near the resources to guarantee the quality and bioavailability of all marketed products at any given time. Just a few months ago, for example, it noted that batches of tetracycline HCl capsules which met official monograph requirements were not bioequivalent to a reference product. As you know, there is substantial literature on this subject affecting many drugs, including such antibiotics as tetracycline and erythromycin. The record on drug recalls and court actions affirms strongly that there are differences among pharmaceutical companies and their products. Research-intensive companies have far better records than those that do no research and may practice minimum quality assurance.

MYTH: Industry favors only "expensive" brand names and denigrates all generics.

FACT: PMA companies make 90 to 95 percent of the drug supply, including, therefore, most of the generics. Drug nomenclature is not the important point; it's the competence of the manufacturer and the integrity of the product that count.

Matters.

MYTH: Generic options almost always exist.

FACT: About 55 percent of prescription drug expenditure is for single-source drugs. This means, of course, that for only 45 percent of such expenditure, is a generic prescribing option available.

MYTH: Generic prescriptions are filled with inexpensive generics, thus saving consumers large sums of money.

FACT: Market data show that you invariably prescribe—and pharmacists dispense—both brand and generically labeled products from known and trusted sources, in the best interest of patients. In most cases the patient receives a proven brand product. Savings from voluntary or mandated generic prescribing are grossly exaggerated.

MYTH: Drugs account for a major portion of the rise in health care costs.

FACT: Drugs represent a very small part of such costs. The amount of the health care dollar spent for prescription drugs was about 12 cents in 1967; today it is about 8 cents. And you as a physician are most conscious of how drug therapy can cut hospitalization, avert surgery, reduce office visits and keep patients on the job.

MYTH: Government intrusions into the marketplace will save tax money.

FACT: Government schemes always cost the taxpayer something, and the costs often exceed the benefits. Certainly, any federal "help," such as lists of wholesale drug prices sent to all physicians and pharmacists, will be no exception. Just think of the expense of keeping them current! Moreover, wholesale prices are poor guides to actual transaction prices and even worse guides to retail prices.

The PMA Position

We believe your freedom to prescribe, either by generic or brand name, should be totally unabridged. Otherwise, your prescribing prerogatives and your relationships with patients will be seriously impaired.

The maker does matter

After the myths about price and equivalency have been shattered, one fact stands out more clearly than ever: *The maker does matter.* As always, your best guide to drug therapy for your patients is to select products—both brands and generics—from manufacturers with credentials and performance records you have come to respect.

PMA

Pharmaceutical Manufacturers Association
1155 Fifteenth Street, N.W.
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SOUTH CAROLINA MEDICAL ASSOCIATION

AUXILIARY

Mrs. James F. Adams, Jr. (Marge)
1519 Kathwood Drive
Columbia, S. C. 29206

Editor's Note: *Following is the Presidential address given by Mrs. Adams upon her installation as President of the SCMA Auxiliary at the recent Convention in Myrtle Beach.*

BROADER PERSPECTIVES FOR OUR SOUTH CAROLINA MEDICAL AUXILIARY 1979-1980

Nellie Claire, honored guests, members of the House of Delegates: I pledge to you and to the talented leaders of this organization who have preceded me that with your help and theirs I will do my best to lead us through another successful year.

I propose to you for this year the idea of broader perspectives in our work. Let's dare to look behind us to see if what we have been doing remains valid. Let's project our medical auxiliaries' activities into the coming several years and try to plan effectively for them. Let's not follow the same path just because it is there, but on the other hand let's not cut a new one just to do something new. Let's be critical and evaluate our work objectively. Through our choice of meaningful projects, let us see to it that our medical auxiliary becomes associated in the minds of the people of our communities with volunteerism and with imaginative leadership which results in positive approaches to problems.

Charles Dickens' first sentence in his "Tale of Two Cities" was: "It was the best of times, it was the worst of times, it was the age of wisdom, it was the age of foolishness, it was the epoch of belief, it was the epoch of incredulity, it was the season of light, it was the season of darkness, it was the spring of hope, it was the winter of despair." This quotation expresses the contradictions which have faced mankind from his beginnings and continue to face us now. The way we mix these opposites will tell the tale of our times, just as it has told the tale of times past.

Let our medical auxiliary through its positive, well-thought-out approach to today's problems have an impact on our times that will help to make it "the age of wisdom, the epoch of belief, the season of light, and the spring of hope."

Mrs. James. F. Adams (Marge)
President

Editorials

The following remarks are taken from Dr. Leon Banov's address to the First Annual Meeting of the Waring Library Society, Charleston, March 9, 1979. It was felt fitting to publish the address by Dr. Gifford to the Society in this issue of The Journal, *and to include in this issue two other articles pertinent to the medical history of our state.*

The Waring Library Society, under the leadership of Dr. Banov and now Dr. Hope Sandifer, has prospered; it deserves our continued support.

— *CSB*

THE WARING LIBRARY SOCIETY: A REALITY

On behalf of the officers and executive committee I welcome you to the first meeting of the Waring Library Society. For all of us with a sense of history and an awareness of a desire to honor the memory of a man who advanced our history, this initial meeting possesses poignancy and revives memories. It is heartwarming for the officers and committee to see and to greet such a warm and friendly gathering.

You should know how this organization was born. At the January 13, 1977, meeting of the Robert Wilson Medical History Club, Dr. William Knisely introduced the idea of "organizing a group to be known as Friends of the Waring Library in order to develop interest and possible contributions." The matter was warmly received and was to be considered more fully at the next meeting.

At the March 17th meeting, Dr. Joseph Waring reported on the proposal for organizing the Friends of the Waring Library. Dr. Knisely suggested that the Health Sciences Foundation of the Medical University of South Carolina act as the sponsoring body. A committee of Doctors Waring, Forde McIver, and Curtis Worthington and Mr. Warren Sawyer was then appointed to draw up plans for the Waring Library Society.

At the May 5th meeting of the Robert Wilson Medical History Club, Dr. McIver, reporting for that special committee, presented by-laws for the Waring Library Society. It was at that meeting, I was elected president; Dr. S. Hope Sandifer, president-elect; and Dr. Layton McCurdy, secretary-treasurer. Dr. Mitchell Rubin was elected a member of the executive committee to serve with ex-officio members Dr. Joseph Waring, Dr. William Knisely, and Dr. Curtis Worthington.

On May 17, 1977, the executive committee of the Waring Library Society had its first meeting in the office of a psychiatrist, with a proctologist presiding. Since then the committee has been meeting nearly every month not in a psychiatrist's office but at the Waring Historical Library where Dr. Sandifer has carried on Dr. Waring's delightful custom of providing Harvey's sherry.

This Society, operating under the auspices of the Health Sciences Foundation of the Medical University of South Carolina, has for its purpose the development of the Waring Historical Library as a major bibliographical research center in the history of medicine and science, and the promotion and perpetuation of an interest in these historical areas. The intellectual and financial strength of this society depends most directly upon the support of you, its devoted and loyal members — that is the key.

As a result of last year's letter writing with an invitation to membership and enlistment of financial support, the Waring Library Society today has a membership of 285 which includes four life members and 38 student members. At present the treasury has $12,961.68.

With its funds, the Society has already started gathering and purchasing books and letters that deal with health care in South Carolina. Moreover, the Society plans to utilize the modern historian's tools: oral history, both audio and audio-visual, and photographs. In effect, the Society wants to capture the drama of the past and the action of the present as they relate to health care in South Carolina.

A study of history should not take place in a vacuum. The aim should be to make people aware of their past. Medical history is not for a chosen few but for everyone.

I want to express my appreciation and thanks for loyal dedicated services on the part of Dr. Hope Sandifer, the president-elect, Dr. Layton McCurdy, secretary-treasurer; the other members of the executive committee: Dr. William Knisely, Dr. Curtis Worthington, Dr. Mitchell Rubin, and Mr. Warren Sawyer, who were elected and Mrs. Anne Donato and Mr. William Matthew who *de facto* were welcomed to the committee by those who were initially elected.

In closing I want to tell of my feelings in a tribute to a man who is not present here this afternoon but who continues to influence me and most of you. I recognize the prized privilege that is mine to have served as the first president of this society, named in honor of a real gentleman and a real scholar and a good friend who taught me both at the College of Charleston and at the Medical University of South Carolina. I have been ever mindful of his example in life for I have found in Joe Waring the inspiration to carry out my duties as president with enthusiasm, devotion, and a dedication for excellence. I thank you for this privilege.

— Leon Banov, Jr., M.D.
103 Rutledge Avenue
Charleston, S. C. 29401

MEDICAL HISTORY IN SOUTH CAROLINA: THE STATE OF THE ART

With Joseph I. Waring's death in 1977, medical history lost a highly respected figure, and scholars working in the history of South Carolina medicine lost surely their leading spokesman. But happily — and Joe Waring would be especially happy about this — the field he nurtured remains strong and viable, and this vitality is due in large part to our Charleston colleague, himself, who did so much to stimulate a like interest in other scholars (this writer among them).

To gauge the vigor of current efforts in medical history, the writer undertook some weeks ago to survey work in progress in the state. This inquiry revealed, for one thing, that practicing and academic physicians continue to do serious research in the history of medicine. Falling mainly in the category of internal history, their writing is of considerable importance because, as inside observers, physicians are in position to tell us a great deal about the evolution of medical science and practice. Among the active physicians is William Weston, Jr. of Columbia, who is currently writing a paper on the work of his father, a leading S. C. nutritionist and pediatrician earlier in this century. In Charleston, the 40-odd physicians in the Robert Wilson Medical History Club also continue active. Club secretary Leon Banov is at work on the history of the management of anorectal diseases. A number of projects are also underway at MUSC: Professors Goldberg and Lipowski, for example, visitors in the Department of Psychiatry, are doing researches in the history of their discipline.

The USC Medical School numbers active medical historians among its faculty, as well. After several years away from historical writing, Charles Bryan, a former recipient of the William Osler prize, has just completed an analysis of 20th Century medical writing by South Carolina doctors. One of the most productive scholars at the Columbia school is Edwin R. Wallace, assistant professor of neuro-psychiatry. Wallace, who followed up his M.D. degree with degrees in history and the history of medicine, has concentrated for years on Sigmund Freud's intellectual history and currently has a book in press entitled, *Freud and Anthropology*.

A most heartening finding of the survey, from this writer's perspective, was the increasing involvement of our state's professional historians with medical history. A decade ago one would have been hard-put to find any South Carolina historian working in the field. The number now, while not legion, is substantial and growing. This is cause for satisfaction beyond the fact of mere numbers, for the historian, though not usually schooled in science, has much to offer the field. Trained to take a wholistic view of society, the historian seeks to place medical development in social and historical perspective and thus provide the external view of medical history to complement the internal perspective of the physician.

One of the most productive of South Carolina scholars is USC historian John P. Dolan. Author of numerous articles (including many in the *Journal*), Dolan and William N. Adams-Smith recently published *Health and Society: A Documentary History of Medicine* (Seabury Press, 1978). Currently, Dolan is at work on a history of medical ethics. At USC's Spartanburg campus, African historian James W. Brown, is interested in the history of epidemic infection and has just contributed a chapter on the disease in colonial Ashanti to *Disease in African History: An Introductory Survey and Case Studies* (Duke University Press, 1978). A co-contributor to the volume, with a study of health and disease in Cameroon, was Mark W. Delancey of USC's Department of Government and International Studies. Another scholar, a specialist in modern European history, is Peter McCandless, of the College of Charleston, who has published articles on the social history of insanity in England. McCandless is currently at work on a book exploring the relationship between insanity and social change in 19th Century Britain.

Much current scholarship centers on the South or South Carolina. This writer, for example, after publishing a number of articles on Anglo-American cooperation in medicine, is writing a social history of health of the Southern poor in the 20th Century. Allan Charles, historian at USC's Union campus and a contributor to the current issue of the *Journal*, is beginning a history of dentistry in South Carolina. M. Foster Farley of Newberry College has just published *An Account of the History of Strangers' Fever in Charleston, 1699-1876* (University Press of America, 1978). Farley's next project will be a history of yellow fever in the state.

As the foregoing suggests, academic historians tend to view the history of medicine as a part of social and intellectual history. Not even much biographical material is represented. One such study, though, has just been published which deserves mention. This is the biography of Dr. Anne A. Young, a pioneer woman physician, who at 87 is still practicing in Anderson. Her story, *Daring Venture*, is the work of Beth Ann Klosky, a professional writer and author, also of Anderson.

As more historians develop specialties in the history of medicine, inevitably more graduate students will be attracted to the field, creating even brighter prospects for medical history. Already a number of such students are appearing. Suzanne Linder, a published historian living in Bennettsville, is writing a medical history of Marlboro County which she hopes to make the basis for a Ph.D. dissertation at USC. Beverly Crosby, another USC graduate student, who has just completed a paper on smallpox inoculation in 18th Century Charleston, is likewise interested in a medical history specialty.

When such students appear they will need to have access to good manuscript sources. In this area much remains to be done. Many rich manuscript collections are still in the hands of owners or their heirs, and if these papers are not acquired soon they will become scattered or lost to other states' libraries. Notably absent from South Carolina archives are the papers of Negro health and medical leaders, such as Dr. L. W. Long of Union and Modjeska Simkins of Columbia, both of whom played lead roles in the Negro health field. Fortunately, however, we have at least two libraries which are eager to acquire medical materials and have the potential to become major repositories. One is the Waring Historical Library, on the MUSC campus, which has abundant material on the operation of MUSC, a large collection of 18th and 19th Century medical student notes (from U. S. and foreign schools), and personal papers of South Carolina professors and practitioners. The other repository is the archives of the South Caroliniana Library (Columbia), whose growing body of medical material includes military surgeons' diaries, the papers of a Greenville doctor, T. Dechard Guess, and various other collections with sizeable bodies of medical material (e.g., the Wil Lou Gray and Mrs. Paul H. Leonard papers). Like the six characters in search of an author, this material needs the attention of medical historians. Given the growing vitality of medical history, we may hope that it will soon receive it.

— Edward H. Beardsley
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CLINICAL USE OF CARCINOEMBRYONIC ANTIGEN IN THE DETECTION OF PRIMARY AND RECURRENT COLORECTAL CARCINOMA*

L. F. KNOEPP, JR., M.D.
J. H. McCULLOCH, M.D.
R. L. CONN, M.D.

Carcinoembryonic antigen, first isolated by Gold and Freedman in 1965,[1] is found in the serum of patients with colon and other digestive tract malignancies. However, it is neither sensitive nor specific. Elevated values are found in patients with other carcinomas, plus a variety of non-malignant conditions, particularly liver disease, cirrhosis, ulcerative colitis, pulmonary emphysema, and patients who smoke.[2, 3] In addition, many patients with primary or recurrent colorectal carcinoma have normal values.[2, 4] Because of this, some authors have despaired of using it clinically and have recommended against its routine use.[4]

Other authors[5, 6] have noted that serial post operative CEA assessment may show, by a rise in values, the first evidence of recurrent disease, and that appropriate surgical intervention may result in some cures in these early cases. This paper presents eight patients in whom a rise in CEA was the first and or only evidence of primary or recurrent colon carcinoma.

PATIENTS AND METHODS

CEA samples were collected with a variety of schedules in this series, due to the lack of a consensus on its optimum use. In general, CEA's were drawn on all patients preoperatively before surgery for colon cancer. Postoperative CEA's were drawn along with a SMA-12 at the time of a physical exam primarily on Dukes C patients as they would be expected to have a greater chance of recurrence. As CEA's became elevated, to greater than 2.5, they were repeated. Confirmed values greater than 5.0 were an indication for non-invasive workup. A consistent or rising CEA greater than 10 in the face of a negative non-invasive workup was used as an indication for a second look procedure.

RESULTS

Results are depicted in Table 1. Eight patients were found, because of an elevated CEA, to have recurrent or primary colorectal carcinoma. No false negative second look operations occurred in this series.

The patients are listed in order of the highest

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* From the Wallace Wilson Brailsford Clinic, P.A., 1776 Skylyn Drive, P. O. Box 2768, Spartanburg, S. C. 29302.

TABLE 1

SECOND LOOK PROCEDURES RANKED BY CEA LEVEL

Patient	1st Surgery	Highest CEA	Pathology	2nd Surgery
1. ES	lt colon	220	liver mets	biopsy
2. MC	rt colon	132	liver mets	biopsy
3. OS	ant colon	100	solitary liver met	resection
4. AF	lt colon	17.8	liver mets	biopsy
5. GC	———	15.7	sigmoid colon carcinoma	resection
6. MM	trans colon	13.2	villous tumor splenic flexure (ca-in-situ)	resection
7. CR	(1) APR (2) trans colon	9.6	peritoneal mets	biopsy
8. OM	rt colon	9.3	local recurrance	resection

CEA value. It can be seen that the four highest CEA values were found in patients with metastatic liver disease.

Decision to perform surgery was difficult when the only abnormality was a high CEA. This occurred in the case of patients 1, 2, 3, and 4. Patients 5, 6, 7, and 8 all subsequently developed some other evidence of malignancy besides the high CEA. In patient 5, a pelvic mass was found and a colonoscopy revealed carcinoma. In patient 6, a colonoscopy showed a villous tumor of the splenic flexure. In both patients multiple barium enemas were negative, but the high CEA values prompted a more elaborate workup. In patient 7, a partial small bowel obstruction developed, for which surgery was needed. In patient 8, a right lower abdominal mass developed after the CEA became elevated.

Results of the "second look operation" are also depicted in Table 1. In four patients a complete surgical resection was possible. Three of the patients are alive and well. This includes patient 3 with a solitary liver metastasis, who is two years post left lateral hepatic segmentectomy, and patients 5 and 6, who had primary colon carcinomas. Patient 8 had a local recurrence resected, but later died of pulmonary metastases. The four patients who had only a biopsy of recurrent disease are either dead of disease (2) or are alive with persistent disease receiving chemotherapy (2).

DISCUSSION

CEA assessment adds another dimension to the diagnosis of primary and recurrent colon carcinoma. An elevated CEA may be the first or even the only evidence of colon carcinoma. It must be remembered both that normal results do not rule out malignancy and that interpretation of moderately elevated values is difficult.

All patients with colon carcinoma should have a preoperative CEA. If the preoperative CEA is elevated, an early (1-2 week) postoperative CEA showing a fall to normal is a good prognostic sign. Follow up CEA's can be done at intervals of every three months, along with physical examination, for two years; then every six months for one more year, then yearly. A CEA less than five is probably normal. Minor variations can be tested for significance with a nomogram as described by Martin.[5] A CEA above five should be repeated to observe the trend. A rising trend is an ominous sign and should prompt a more complete evaluation of the patient, including SMA-12, liver scan (possibly CT scanning), barium enema, sigmoidoscopy, and possibly colonoscopy. Even in the absence of a confirmatory non-invasive workup, a rising CEA trend in our judgement warrants a second look exploration. If metastatic disease is found which is not resectable for cure then at least an opportunity for debulking surgery will exist and chemotherapy may be started earlier with perhaps better response. As shown in this small series of 8 (Table 1), some patients will be found to have treatable or possibly curable disease, as in patients 3, 6, and 8.

The use of CEA analysis in patients without a history of colon carcinoma or gastrointestinal malignancy (as in patient 5) has not been supported by the literature; at present we do not recommend it as a routine.

Martin, *et. al.*[5] performed second look operations on 25 patients who had elevated CEA values, based on a CEA nomogram. Criteria for re-exploration was a two standard deviation elevation of CEA, confirmed on a second sample; 22 of 25 patients explored had recurrent tumors. Six had localized recurrence, 16 had metastatic disease, two had second primary (resectable) gastric carcinomas, one had a resectable carcinoma of the Ampulla of Vater. The six patients with a resected recurrence are all doing well without elevated CEA's.

Sugarbaker, *et. al.*[6] followed 33 patients with resected colorectal cancer by CEA's. Recurrent disease was found by physical findings in five patients, by rising CEA titers in four others, by a combination in two and by surgical exploration in one. CEA's were done monthly, and other tests and clinical examination at three to six month intervals. Their feeling was that although CEA assay could not replace clinical exam, it might identify patients needing chemotherapy earlier or it might identify patients for whom a second look procedure could be done.

SUMMARY

High CEA titer was demonstrated to document (as the first sign of any abnormality) the presence of colorectal carcinoma in eight patients. One patient had primary carcinoma of the colon, seven others had recurrent or new primary lesions after previous colon resection for cancer. In four patients, all with carcinoma metastatic to the liver, a high CEA titer was the only preoperative test positive.

Three patients are alive and free of disease at this time and two patients are alive under treatment, perhaps because a rising CEA allowed the earlier diagnosis of cancer.

CEA assay is felt to be a useful adjunct in following patients with previous colorectal cancer. □

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THE MANAGEMENT OF PEPTIC ULCER

VINCE MOSELEY, M.D.*

Peptic ulcer, or, as some term it, acid peptic disease, is manifested by the acute development, healing and recurrence of more often single but occasionally multiple, punched out lesions involving the alimentary tract mucosa in a discrete manner with adjacent mucosal inflammation. At times, the ulceration may involve the submucosal and muscle structures with actual perforation, but usually healing occurs with little residual scarring of a significant degree, though at times cicatricial deformity and obstruction may occur both in the antral area of the stomach and duodenal pyloric area. These ulcers occur in those areas of the gastrointestinal tract which are exposed to acid and pepsin, namely the distal esophagus, stomach, duodenum and ileum in association with Meckel's diverticulum with functioning gastric glands, or in jejunum or ileum after gastroenterostomy or gastric perforations into a segment of intestine. An earlier dictum of "no acid, no ulcer" still holds. This is in contradistinction to "acute ulcers" associated with severe infections, trauma, burns, intracranial injuries or shock where ulcerations associated with diffuse mucosal injury may arise at any area of the gastrointestinal tract and not just those where acid-pepsin exposure occurs. These latter forms of ulcer often rapidly perforate.

Although there are several distinct differences in respect to the peptic ulcers involving the stomach and those of the duodenum, currently our approach to therapy and understanding of some aspects of the pathogenesis of these is similar enough for us to group gastric and duodenal ulcer in the same disease category of acid pepsin disease.

Although acid and pepsin are essential pathogenetic elements, there is considerable doubt that the disorder relates to an excess of these. Although individuals with duodenal ulcer and other members of their family group are found to have increased amounts of hydrochloric acid with maximal "Histalog" stimulation or with pentagastrin when compared with those not prone to ulcer disease and have about 75 percent more functioning parietal and oxyntic cells, they also have greater gastric mucosal surface area and volume. This probably also accounts for the finding of increased levels of plasma pepsinogen also observed in such individuals and their family members. In gastric ulcer, however, except when it occurs after many episodes of duodenal ulcer with partial obstruction of the duodenal or pyloric channel, it is not so associated with increased gastric mucosal surface area, nor is it associated with another interesting finding of lack of secretion of the A, B, and AB factors in the saliva or intestinal secretion or with frequency of being associated with O blood type and certain HLA tissue types as is duodenal ulcer.

The discreteness of the ulcerations suggests that some ischemic reaction involving the mucosal blood supply or its submucosal arteriovenous anastomotic plexuses occurs. The vascular changes associated with emotional, toxic, mechanical, chemical, endocrine or circulatory engorgement on the venous side certainly is to be, at times, implicated. As examples we know that cirrhosis or portal hypertension of other cause and right heart failure are often accompanied by peptic ulcer, as are states of hyperadrenocorticism. Also ischemia and embolism may give rise to ulcers, and the frequency of peptic ulcer in polycythemia is thought to have such a mechanism or one of thrombotic origin. Also chronic lung disease is associated with peptic ulcer. In some instances allergy appears to be the inciting factor, with capillary leakage and local edema.

The classical observations of Beaumont on Alexis St. Martin and of others with the opportunity to observe patients with a gastrostomy, such as Wolf and Wolfe's observations on "Tom," have provided much grounds for speculation as to the relationship of emotional, psychic, physi-

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cal or environmental stresses in peptic ulcer causation. Spring and fall or early winter climatic changes seem to have more than a casual relationship for many in respect to periods of recrudescence, but the universal applicability of these observations or other psychosomatic theories of causation remain more selectively than universally applicable. Rest of a physical or mental type yet remains as well as attempts to assist the individual in better methods of coping with interpersonal, employment, or other external problems, as important aspects of management, both in acute and long-term situations.

Most authorities now agree that there is not, as such, a type of personality which is ulcer prone, and don't we all have problems of coping and recurring interpersonal relationship problems at home and elsewhere of varying degrees of duration?

A recent report by Dr. J. I. Isenberg of Wadsworth Hospital Center, a VA Hospital in Los Angeles, provided data to indicate that although very prevalent, that peptic ulcer disease has been decreasing for the past 30 years despite the figures that annually some 400,000 persons require hospitalization, and some 7,000 die. Males are fives times as often affected as females. Currently 10 percent of all males and 5 percent of all females may anticipate this disorder during their lifetime. Annual medical costs are some 1.5 to 2 billion dollars.

These and other aspects of the acid-peptic theory as to causation have led us to develop certain rationales that have been incorporated into our therapeutic regimens and management programs. Measures that have been accepted without much question in previous years, such as special diets, antiacids, anticholinergics, rest, mild sedation, and in some instances varying degrees of psychotherapy, have in the past several years been subjected to increasing scrutiny and are now the subject of considerable debate and are considered by some to be of extremely dubious benefit. To quote as an extreme position, Dr. Eddy D. Palmer in the 2nd Edition of *Practical Points in Gastroenterology*, "Ulcer disease is a whole body disease. The patient has trouble with people and it, the ulcer, is caused by people. Don't hospitalize the patient unless there are complications." He recommends no diet, no drugs, no restrictions, but only psychotherapy.

This is at variance with the belief of most gastroenterologists who although recognizing that ulcers may often heal without specific therapeutic regimens, have also had the disappointing experience associated with the tremendous enthusiasm and acceptance existent in the late 40's and 50's for psychophysiologic or psychosomatic diagnoses with later disappointments in this as an etiologic or therapeutic approach.

What firm areas of knowledge do we now possess in respect to gastric physiology and the application of these in our treatment programs?

To recall these to mind, let us review briefly some of what we know about gastric motility and secretory functions.

Gastric motility is activated by vagal stimulation and the effects of acetyl choline release at the myenteric plexus level, and this is influenced by cephalic stimuli originating at higher and medullary brain levels. At the myenteric levels, in addition to the vagus, various stimuli are produced by food and other ingested substances, and their effects on the secretion of the various humeral factors by the glands of the gastric, duodenal and small intestinal mucosa of both stimulatory and inhibitory types. Relaxation of muscular contractions is also induced and modified by the sympathetic nerve pathways and their plexuses. Through these pathways are also transmitted painful sensations to the dorsal ganglia and spinothalamic tracts. Ulcer pain is a result of smooth muscle contraction. The mucosa has no pain receptors of itself. Tenderness and referred pain represent deep pentration and irritation of the parietal peritoneal surfaces and is transmitted as a pain sensation by the musculoskeletal thoracolumbar spinal nerve pathways.

To relieve pain, there is need, therefore, to produce relaxation of the muscular contraction in respect to the visceral gnawing, grinding or burnings sensations described, which typically as sympathetic nerve transmittal pain is felt in a small area towards the midline of the epigastrium. An inquiry as to what type of discomfort is felt, where it is felt — and have the patient point to the area with the bare abdomen exposed — is necessary, when it is felt, and what increases or decreases the discomfort. All these give necessary diagnostic clues. Other aspects of the pain or associated tenderness, with some muscle guarding or rebound, or referral, will give additional information about the depth of penetration of the lesion and the area of parietal peritoneum thereby irritated or inflamed. It should be pointed out that frequently with posterior wall duodenal ulcer, pain is acutely perceived at the level of the 5th to 8th dorsal spinous processes, and with gastric ulcer the left flank, the left lower thoracic and the left shoulder areas, and that pain with cutaneous hyperesthesia may be present, and muscle guarding or splinting when penetration and parietal peritoneal irritation is present.

Measures then to relieve pain will consist of such things as the appropriate use of vagal and acetylcholine inhibitory medications, such as atropine, hyoscyamine, belladonna or one of the effective synthetic agents with the omission of foods which may be stimulating, such as coffee, meat extracts, alcohol, and the use of those that may be relaxing in effect such as carbohydrates and fats, is a reasonable approach. Frequent small feedings will also serve the purpose of reducing muscular contractions associated with an empty gastric reservoir.

Where we have gone wrong in the past is to believe that a prolonged period of such therapy is required, and that rigid adherence to a dietary program will, in fact, hasten healing or prevent recurrences of an ulcer. It is believed to have been shown in a number of studies that even in those patients who are regularly prone to seasonal relapses in the late fall or early winter, or in the late winter or early spring months, that strict diets have no proven prophylactic effectiveness.

What is the role of antiacids in the relief of pain? Here we are not certain, except for perhaps some effects in decreasing the release of substances of a stimulatory type from foods through interference with peptic activity, or by the raising of pH and its effects on the motor functions of the antrum, pylorus, and duodenal bulb. Alkalinization has no effect on ulcer pain per se, nor does lowering of pH as may be shown by dripping 0.1 normal hydrochloric acid on an ulcer crater produce increased pain, but with intravenous atropine or hyoscymine will provide immediate relief if pain is present.

When, then, should we believe that alkalinization is needed or is effective in the healing of an ulcer?

Based on our current knowledge, we believe that an ulcer occurs in an area which has lost its defenses against autodigestion by activated pepsin, and that hydrochloric acid in a concentration sufficient to produce a pH of between 1 and 3 is required for the precursor of pepsin, namely pepsinogen, to be activated into its active enzyme form. Elevation of the pH by reducing hydrochloric acid formation or neutralization of that formed so that the gastric pH becomes at a level of 3.5 or above, will effectively prevent pepsin digestion, and will stop the transformation of pepsinogen to pepsin.

As with the previously described activities and the areas where stimulating or inhibitory functions occur in respect to the vagus nerve, the myenteric plexus and the sympathetic nervous system, these same sites are involved in respect to the production of hydrochloric acid in the parietal cells, of pepsinogen in the oxyntic cells and mucous and slightly alkaline gastric juice by the neck and mucous cells of the gastric glands and mucosal surface. Thus excessive and prolonged acid production may relate to cephalic or brain area stimulation, mucosal — particularly antral stimulation with gastrin release, and duodenal and small intestinal release of inhibitory and stimulating humeral substances.

Thus certain foods such as proteins stimulate the antrum to produce gastrin and stimulate hydrochloric acid and pepsin secretion, and though initially as amphoteric substances may buffer and partially neutralize the acid over time, the buffering effect is lost and the stimulatory effect supervenes. This is true also of the effects of protein later when in contact with the ileum. Table A illustrates this effectively. Fats through their effects on the duodenal mucosa will stimulate the release of secretin and cholecystokin which, though more directly related to stimulatory activities in regards to stimulating the secretions of the pancreas by secretin with a flooding of the duodenum with an alkaline fluid rich in bicarbonate, as well as lipase and some trypsin, and

TABLE A

Average Gastric pH Values After Administration of 100 cc. of Milk and Cream and After 100 cc. of 15 gm. Protein Mixture

Fasting	15 min.	30 min.	60 min.	11/2 hr.	2 hr.	3 hr.
4	*5.5	4.5	4	3	1.5	1.5
4	+6	5	4.5	4	2	1

*Milk and Cream
+Protein Powder Mixture

The continuing drop in pH at 2 and 3 hours is believed due to protein digestion and stimulation of gastrin release i.e. the "Intestinal Phase" of acid secretory stimulation.

contraction and emptying of the gall bladder by cholecystokinin, both of these humeral factors also reduce gastric secretary and motor function in higher concentrations.

Thus we can understand why foods and anticholinergics may provide beneficial effects by establishing conditions where painful muscular contraction may be inhibited or lessened, and also play some role in the lessening of further insult by acid activated pepsin in the promotion of ulceration and the inhibition of healing.

Can hydrochloric acid per se cause mucosal injury and ulceration? In its usual physiological concentration of 0.16 normal, it does not, unless there is a loss of mucosal barrier and there is back diffusion. The mucosal cell barrier may be broken by higher concentration of hydrochloric acid by salicylates, by alcohol in excess of 10 percent concentration, by bile acids with lysolecithin, various detergents and by colchicine, Indocin and a number of other medications. Excessive histamine and serotonin stimulation, elevated blood calcium, and adrenal gluco-corticoids also in some manner may disrupt the mucosal barrier and allow backward diffusion of HC1.

At this point we should point out that the mucosal barrier is not mucous but a lack of permeability of physiologic strength HC1 through the intact mucosa. Mucous can bind hydrogen ions but not in significant degree, and has no role in the mucosal barrier per se. When the barrier is broken, the back diffusion of the gastric HC1 penetrates the mucosa.

What the mechanisms are which render the mucosa susceptible to ulceration in such situations as in the Zollinger-Ellison and polyadenomatous conditions is not known, nor why this is true in cirrhosis, pancreatitis, polycythemia, rheumatoid arthritis, chronic obstructive pulmonary disease, hyper-parathyroidism and hyper-adrenocorticism. Hypoglycemia and increased serum calcium will both stimulate gastric secretion but do not produce of themselves a break in the mucosal barrier. In rheumatoid arthritis, vasculitis is implicated.

How effective is the administration of antiacid in the control of pH and thus of pepsin? Most of those which are used in different formulations, including bicarbonate of soda, calcium carbonate, magnesium oxide, aluminum hydroxide and magnesium trisilicate in adequate quantity will effectively raise the pH to a level of 3.5 to 5, but their neutralizing efficacy is more demonstrable as are those of the various protein preparations, including milk in vitro than in vivo as shown in Table B. Actually as can be demonstrated by periodic sampling with a gastric tube or by the use of Heidelberg capsule with a radio transmitter to detect constant pH changes with radio monitoring, the effects of most antiacids as buffering agents last only about 40 minutes. Continuing secretion and rapid emptying the stomach of the administered antiacid nullify its effects rather rapidly. Many believe the stimulus to gastric emptying and relaxation of the tonus of the pylorus and duodenum are actually the mechanisms for pain relief rather than acid neutralization. Bismuth aluminate has actually little neutralizing effect in its usual dosage, but it is quite effective in ulcer pain relief of gastric pyloric or antral ulcers. Pepsin is effectively bound and inactivated by bismuth. Except in the initial stages of ulcer management, when there is much pain or distress, antiacids are best administered at 1 to 2-hour intervals after a meal. The food provides initially adequate buffering, which, as gastic emptying occurs and continued acid and pepsin production continues by stimuli from the small intestine, then the antiacid exerts its beneficial effects. Administration at bedtime and if distress should occur at 1 to 2 A.M., a repeat dose is given.

TABLE B

Average pH values 8 subjects given 100 cc. of Milk and Cream every hour. pH reading taken fasting and at hourly intervals just prior to readministration of milk and cream, and repeat with alternate antiacids and antiacid alone

Fasting		1 Hour		2 Hours	3 Hours	
4.2	M&C	3.4	M&C	2.8	M&C	2
5.0	M&C + alk.	5.6	M&C alone	1.8	M&C + Alk.	5.3
5.0	Alk. No M&C	6	-	3	-	2

Data suggest best pH control with feeding plus antiacid at hourly intervals.

For the more acute stage of treatment, hourly administration will usually be necessary, along with frequent small feedings on the half hour of an hourly program. In most instances after four to six days, a three-meal program with post-meal antiacid will prove adequate. If for additional need an anticholinergic should be required, this should be given before meals for adequate absorption and not with the aluminum or trisilicate preparations which will adsorb and prevent absorption of an anticholinergic alkaloid.

Although effective and with rapid relief, absorbable sodium or calcium antiacids should be used only briefly because of the real dangers of producing systemic alkalosis, the milk alkali syndrome and renal damage, including calcium calculi.

As to which of the many preparations to use, I think this is chiefly a matter of patient acceptability as to taste, texture, appearance and whether in the doses employed it produces diarrhea or constipation. In long-term use in the presence of kidney damage, magnesium retention at toxic levels may occur, and also phosphorus depletion with the aluminum salts may occur, if phosphate intake from diet restriction should happen, which is extremely rare. Cost is also a factor and may vary from a few cents to 35 cents per dose.

In brief, then, our objectives in the symptomatic management and treatment of an acute or recurrent peptic ulcer is to promote physiologic rest of the stomach, elevate the gastric pH to 3.5 or above to inactivate pepsin, where necessary use anticholinergics to relieve painful muscle spasm and deal with evident precipitating factors as intercurrent infection, physical, nervous, or mental fatigue with loss of sleep and overt or cryptic anxiety where this may be discovered.

Recently, after many years of trial in England and Europe, "Cimetidine," or "Tagamet" has become available as an effective agent to block the histamine H-2 receptor sites, and thus effectively diminish acid production as a result of histamine, gastrin and similar humeral agents produced with food digestion. Many with considerable experience also find that anticholinergics may be added with benefit in the acute stages, and with gastric ulcer antiacids may often also be required. Whether "Cimetidine" is as effective with gastric ulcer in treatment as it is in duodenal ulcer remains uncertain. Also whether it will prove to be safe for long-term administration or as a prophylactic measure for those with regular spring and fall relapses also remains questionable. Other sites where H-2 receptors may be blocked by "Cimetidine" are in the brain, heart, lung and uterus.

Other therapeutic agents which bear promise, particularly for gastric ulcer treatment, are certain pectins, carbenozolone sodium which is derived from licorice and has many of the properties of aldosterone and, in fact, is blocked by aldactone. Sodium and water retention often requires a thiazide diuretic. Also bismuth salts are useful. Anticholinergics may be inadvisable as these delay gastric emptying and relax the pyloric sphincter, allowing bile acid regurgitation. Gastric stasis alone may result in ulcer, as is often seen after duodenal or pyloric scarring and biliary reflux can cause mucosal alterations with acid penetration of the mucosa.

Recently antisecretory prostaglandin E-1 has been introduced for research as a gastric secretory inhibiting agent, and a renewal of interest in the substances gastrone and entero-gastrone and urogastrone, earlier studied by Ivy and thought by some to be the combination of cholecystokinin and secretin, but now believed to be separate, perhaps may bear some promise. Certain polysaccharides are mucous stimulating and are being studied as mucosal protective agents. Of interest is the recent observation that urogastrone or a derivative of enterogastrone, derived from Brunner's glands in the duodenum, is secreted also in quantity by the salivary glands, and has revived interest in very thorough chewing of food as being a rational approach also for ulcer patients to pursue.

Finally, what should our program of follow be? If after six weeks a duodenal ulcer patient is pain free and has been so for some time, most restrictions should be discontinued with some advice as to precipitating factors and the avoidance of certain known medications, including alcohol in concentration in excess of 10 percent, and hopefully the beneficial effects that rest, relaxation, and the intermittent regular use of antiacids may provide under periods of stress or seasonal changes. If the patient has had several episodes, a repeat x-ray after six weeks may be advisable to determine if there is much scarring which might lead later to gastric ulcer. With a gastric ulcer, re-x-ray to be certain of healing completely and if any doubt, gastroscopy and biopsy. Currently evidence is against benign ulcers being precursors of carcinoma, but ulcers may occur in carcinomas and heal.

Will long-term, regular administration of postprandial and bedtime antiacids, along with an anticholinergic, reduce the frequency of relapse? Several years ago, the late Dr. Julian Ruffin of Duke University and Dr. David Cayer did an extensive study, as well as one by Dr. Sun, which indicated that as compared with placebo, there was a significant reduction in ulcer recurrence over a 2 to 10-year period as shown in Table C.

TABLE C

Ruffin and Cayer

10 Year Follow

103 Patients	Placebo -	52% Relapse
226 Patients	Atropine --	47% Relapse
705 Patients	Anticholinergic Synthetic	- 26% Relapse

Other RX PRN Antiacid - Bland Diet

2 Year Follow

Sun

20 Patients -	Placebo	45% Relapse
25 Patients -	Anticholinergic -	8% Relapse

PRN Bland Diet and Antiacids

Any studies of this type must be of considerable duration in time, as peptic ulcer, though recurrent and particularly so in the spring and fall months, also recurs in a longer cycle of about seven years with periods of relapse at intervals of six months to two years, and at other cycles with intervals of freedom of recurrence for period of five or more years. Why is not known.

TABLE D

12 Year Follow of 101 Peptic Ulcer Patients (8 Gastric)

54 Rx PRN with Q3 Months Checkups
- 26 Frequently relapsed - 18 complications, 12 to surgery
- 20 Relapsed intervals of 2 to 5 years
- 8 No relapses

20 Rx Antiacid Tid 2½ hrs. PC and HS, Monthly Visits
- 8 Relapsed in 2 years
- 12 No relapse in 5 to 9 years

27 Rx Antiacids Tid 2½ hrs. PC and HS plus Anticholinergic Tid AC and HS - Monthly Visits
- 24 No relapse in 12 years
- 3 Relapsed: 1 after 11 years, 2 frequent, with 2 requiring surgery

Patients in each group similar in distribution of age and sex. 85% were black.

In Table D, I have shown my practice experience of three groups of patients quite similar in age, sex and economic status. One group was seen periodically and treated on a PRN basis. The second group chosen because of their more frequent relapses was treated with simple dietary restrictions in respect to alcohol, meat stews, gravies and soups, spices and TIDPC and HS use of antiacids, more frequently with colds or intercurrent illness. For the third group, Tr. of Belladonna before meals and at bedtime, along with antiacids 2 to 2½ hours PC and at bedtime was given. As you may see from this study, a real reduction in relapses was observed. No problem as to alkalosis, renal stones or damage or evidence of phosphorus depletion was observed.

When is surgery indicated for ulcer complications? I believe the need for surgery should be limited to the following: free perforation, pyloro-duodenal obstruction by cicatrix, hemorrhage unresponsive to proper management, confined perforations with intractible symptoms, Zollenger-Ellison Syndrome, probable malignancy with suspicious cytology on biopsy of gastric lesion.

What about the frequently relapsing patient? Some real benefits are achieved, but in the majority in my experience I prefer to deal with ulcer pain, controllable hemorrhages, and the various emotional difficulties rather than the problems of dumping syndrome, severe hypoglycemic episodes, diarrhea, afferent and efferent loop syndromes, as well as weight loss, malnutrition, osteoporosis, marginal ulcers and anemia, all problems which arise in varying degrees regardless of whether the surgical procedure is just vagotomy and pyloroplasty, or whether a Billroth I or a Billroth II procedure or simple gastroenterostomy.

It has often been a real problem for me to undertake the care of such patients after their referral by a surgeon at the end of his ropes, who, after much manipulation, eventually refers the patient for medical management because surgery has done all it can. ☐

LIFE STRESS AND HEALTH*

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The public typically views stress as a major risk factor for coronary heart disease (CHD)[1] and for other health problems as well, a view apparently held by many members of the medical profession.[2] Dr. Bryan, in an editorial in the October, 1978, issue of *The Journal*, commented on our lack of attention to stress in the article on CHD risk factors in the same issue.[3] Although not mentioned in our article, the Multiple Risk Factor Intervention Trial (MRFIT) is obtaining data on various stress indices, but reports on this topic are not yet available. My colleagues and I, however, are engaged in another study on which we can report at this time.

The Liberty Corporation Employee Health Study was initiated in 1976 by investigators from the University of South Carolina and Liberty Corporation (LC) in Greenville, South Carolina. The major purposes of the study are to examine the relationships among health behavior, health status, and employment related variables. The specific purpose of this report is to examine the relationships between stressful life events and health status.

METHODS

Headquarters personnel (n=527) at LC were given an examination in the company medical department during 1976-77. After the program had been explained by memo from the Chairman of the Board and in person by one of us (HGH), employees gave their informed consent to participate in the study (only 23 employees refused to participate). Of the 504 participating employees, 190 were male and 314 were female. There were 36 blacks, 467 whites, and 1 classified as other. Most (76%) were married, and 97% had at least a high school education. Hence, the sample can be described as mainly white, relatively young (average age — 34 years), mostly married group of white collar employees.

Measurements. Although many clinical, physiological, psychosocial, and employment related variables were obtained, this report will be primarily limited to three; stressful life events (LE), Cornell Medical Index (CMI), and body (BC) and self (SC) cathexis. The LE check list contained 143 items representing seven categories of stressful life events: (1) deaths, (2) separation, (3) arguments with important persons, (4) major threats to self or self-image, (5) minor threats to self or self-image, (6) threats to material well-being, and (7) residual life changes. Each item has been assigned a weight corresponding to the relative seriousness of the event. Recency of the event was also considered, study participants checked events as occurring in the last year (LELY), one to two years ago (LE12), occurring three or more years ago (LE3), life events ever (LEE) and total life events (LET). The weights

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for checked items were summed to give a life events score for each person for each of the time categories. Test-retest reliability for this procedure has been reported to be r=.81.[4]

The measure of health examined for this report was the CMI.[5] In this self-administered questionnaire, an individual checks whether or not he experienced symptoms described in 195 probes related to various organ systems, selected diseases, frequency of illness, feelings and mood. Although CMI is not sensitive enough to be a reliable diagnostic tool for specific diseases, it does indicate the presence of emotional ill-health and serves as an index to general overall health.[6] The CMI has been widely used in epidemiological studies, and has been shown to have acceptable validity.[6]

A third item of interest was body-cathexis (BC) and self-cathexis (SC). BC refers to the degree of satisfaction or dissatisfaction with the body or its parts; BC may also be thought of as body image and SC as self concept. BC is strongly related (r=.6) to SC. These variables were measured by questionnaire using the procedure of Secord and Jourard.[7] This scale contains a list of 46 body parts and functions and 55 items on conceptual aspects of self. The participant is asked to assign a number best representing their feelings about each item according to the following scale:

(1) Have strong feelings and wish change could somehow be made.
(2) Don't like, but can put up with.
(3) Have no particular feelings one way or the other.
(4) Am satisfied.
(5) Consider myself fortunate.

A total score was obtained by summing the ratings for the 46 items for BC and 55 items for SC. The split-half reliability for BC has been reported to be r=.81 and for SC to be r=.90.[7]

Data Processing. The questionnaires were edited for completeness in the LC Medical Department, then keypunched and verified in the Keypunch Department. The raw data tapes were then sent to one of us (AB) at the National Cancer Institute, where numerous editing procedures were implemented. Errors were referred back to the LC Medical Department for correction and/or ajudication. Summary variables were created and the tapes readied for data analysis. Data for this report were analyzed on an IBM 370/168 computer using SPSS programs for Pearson correlation and analysis of covariance (ANCOVA).

Results. The initial data analysis procedure was to examine relationships among CMI, LE, BC, and several other variables thought to be potentially confounding. Pearson correlation coefficients are shown in Table 1. The relationship between CMI and LELY was highly significant (r=0.40) and further analyses to control for confounding seemed justified.

CMI, the dependent variable, was analyzed for the effects of independent variables (LELY,

Table 1. Pearson Correlation Matrix for CMI, LE, BC, and Selected Confounding Variable (N=502)

Variable	CMI	#CIG	HK	BC	LELY	LE12	LE3	LEE	LET	AGE	SEX	MS	SCHYR	SC
CMI	1.00													
#CIG	.05	1.00												
HK	-.17	-.02	1.00											
BC	-.37	-.02	.01	1.00										
LELY	.40	-.03	-.03	-.08	1.00									
LE12	.03	.02	.02	.01	.19	1.00								
LE3	.01	.06	.06	.02	.11	.59	1.00							
LEE	.24	.01	.01	-.08	.40	.11	.02	1.00						
LET	.37	.14	-.01	-.07	.93	.51	.36	.50	1.00					
AGE	-.15	.13	.32	.01	-.22	-.02	.12	-.13	-.19	1.00				
SEX	.37	-.16	-.32	-.31	.08	.03	-.07	.07	.07	-.40	1.00			
MS	.07	.02	-.17	-.02	.11	-.02	-.04	-.03	.07	-.18	.18	1.00		
SCHYR	-.32	.06	.38	.17	.03	.03	.09	-.03	.04	.17	-.59	-.03	1.00	
SC	-.37	.13	-.02	.64	-.13	.03	.00	-.12	-.11	.04	-.25	-.05	.20	1.00

#CIG=cigarettes smoked/day, HK=score on health knowledge test, MS=marital status, SCHYR=number of years of schooling

With N=500, all correlations of .09 or greater are significant at p=.05

age and sex) after controlling for cigarettes smoked (#CIG), education (SCHYR), health knowledge score (HK), BC and SC. LELY categories were based on dividing the group into thirds based on LELY scores. Age categories were ≤25 years, 26-39 years, and ≥40 years. The ANCOVA procedure first adjusted for the covariates then computed main effects for the independent variables. The results of this analysis are presented in Table 2. All covariate effects were statistically significant. The main effects for LELY and SEX were highly statistically significant, while the main effect for AGE was not. The magnitude of the significant effects are shown in Table 3. The mean and standard deviation of CMI for the entire group were 18.70 and 13.63, respectively.

Table 2. ANCOVA for CMI by LELY, SEX, and AGE with #CIG, SCHYR, BC, SC, and HK as Covariates (N=502)

Source of Variation	df	Mean Square	F	Significance of F
Covariates	5	4493.9	37.37	.000
#CIG	1	669.6	5.57	.019
SCHYR	1	3247.9	27.02	.000
BC	1	1952.4	16.24	.000
SC	1	2554.5	21.25	.000
HK	1	700.9	5.83	.016
Main Effects	5	2147.1	17.86	.000
LELY	2	4351.5	36.20	.000
SEX	1	1506.4	12.53	.000
AGE	2	185.3	1.54	.215
2-Way Interactions	8	198.3	1.65	.108
LELY x SEX	2	370.9	3.09	.047
LELY x AGE	4	209.9	1.75	.139
SEX x Age	2	17.7	0.15	.863
3-Way Interactions				
LELY x SEX x AGE	4	189.4	1.58	.180
Explained	22	1615.7	13.44	.000
Residual	479	120.2		

To further examine these relationships, participants were divided on the basis of sick leave (SLH) (i.e., ≤two days >two days). LELY was postively associated with CMI in each SLH category. There were no important differences found between these two analyses and those reported in Table 2. The main effects for LELY and SEX (when adjusted) were essentially the same as those for the entire group (Table 3).

A stepwise multiple regression analysis (with CMI regressed on all variables listed in Table 1) identified three variables that comprised the best model for predicting CMI. These variables, in order of the magnitude of their contribution to the equation, were LELY, BC, and SCHYR. BC and SCHYR carried a negative sign, indicating an inverse association with CMI. The multiple R for these three variables and CMI was .595, the adjusted R^2 was .354. The addition of other variables to the model did not significantly improve predictive ability.

Table 3. Adjusted Effects of LELY and SEX on CMI.

Categories	Mean CMI Adjusted for all Independent Variables and Covariates
LELY	
Lower third	13.71
Middle third	18.19
Upper third	24.46
SEX	
Male	15.67
Female	20.61

Discussion. The primary purpose of this paper is to examine the relationship between life event stress and general health status in an employee population. This type of research is fraught with many difficulties and controversies. The term "stress" itself has been much maligned and subject to misinterpretation.[8] Selye maintains, however, that it is a useful term with specific meaning.[9] Many will agree that certain events or activities can be identified as stressors and further that these stressors can have impact on various aspects of human health. In this report we are concerned with one type of stressor, significant life events, and its impact on one measure of health as determined by the CMI.

Our finding that individuals who reported higher frequencies of stressful life events had poorer scores on the CMI is consistent with most,[10] but not all reports.[11, 12] Rahe has suggested that psychological stress can both predispose and precipitate physical illness.[13] We found that CMI scores were significantly correlated with LELY after adjustment for # CIG, SCHYR, BC, SC, HK and sex indicating that individuals with high LELY scores are susceptible to poorer health status (high CMI). Multiple regression analysis showed LELY to be the most important predictor of CMI, with SCHYR and BC also entering the equation.

Stress research is fraught with methodological controversies.[14, 15] For example, it could be suggested that the CMI-LELY relationship results because these scales measure similar contructs. The non-significant correlations for CMI with life events from earlier time periods (LE12 and LE3) and the lower correlations for CMI with LEE and LET argue against that point. Likewise, the relatively low correlations between LELY with LE12 and LE3 support the idea that different constructs were being measured. The increasing correlations between LELY with LEE and LET were expected since overlapping constructs were included.

Another explanation could be that individuals in ill health experience more life events and score higher on the CMI scale. The persistence of the CMI-LELY association in high and low SLH groups, however, suggests that the high LELY and CMI association is not due to sickness. This is consistent with the idea that the CMI measures a general health variable and not illness specifically.

While it would be unwise to read too much into the results of this cross-sectional study, the following tentative conclusions seem warranted.

1. This report, and many others, suggest that individuals subjected to elevated life events stress may be more likely to experience less than optimal health status.

2. Other social or personal characteristics such as educational level and body image must also be considered in any model of health and life event stress.

What is the status of life event stress research in terms of guidelines for the practicing physician? Although Rahe suggests that the patient's life stress load should be elevated, he provides no specific recommendations for utilization and follow-up.[13] One clue may come from studies suggesting that certain intervening variables may be important in helping individuals deal with various types of stressors by providing support and reducing the impact of the stressor.[9, 16] For instance, Nuckolls and colleagues found that pregnant women with high psychosocial assets had fewer complications than those with low assets.[17] Howard and colleagues found that employees varied in the effectiveness with which they coped with job related stress.[18]

In summary, we believe that life event stress is likely to have an impact on health. This effect may range from morbidity or mortality such as due to coronary heart disease, to less serious but important factors such as monitored in this study. Although many of the mechanisms need to be further explored and elucidated, various types of support from friends, family, significant others, and personal resources can help reduce the impact of these stressors. The wise physician may help by identifying patients at risk and helping them focus on existing sources of support and/or help establish new ones. □

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1979 PRESIDENTIAL INAUGURAL ADDRESS

HARRISON L. PEEPLES, M.D.*

The Journal *is pleased and proud to publish below the text of Dr. Peeples' Inaugural Address, delivered in Myrtle Beach, South Carolina, Saturday, April 28, 1979, on the occasion of his inauguration as the 115th President of the South Carolina Medical Association.*

Mr. Chairman, members of Council, Presidents of Component Medical Societies, Guests, Ladies and Gentlemen —

I accept this honor and responsibility with a pledge to you to devote my best effort to represent you and your policies to the public and to our membership.

It is indeed a pleasure to speak to you. This evening I shall attempt to give you the broad picture of organized medicine and the profession as I see it. I shall speak to you about our relationship one to another, our relationship to our patients, and our relationship to our government. My suggestions to you as to the specifics of what we will be doing and what our goals should be will be conveyed to you through the President's Page in the SCMA *Journal* and the Newsletters during the year. With your invitation, the officers and councilors would welcome opportunities to visit with you in the local medical societies.

Dr. Hawk has told you of our organization's accomplishments last year. I would commend Dr. Hawk on his leadership and would commend the Council and its Chairman on their devotion to duty and to the attention to general affairs and the internal operation of the organization. Our Executive Director has gathered an excellent staff and they are available to assist the membership. I commend him and the entire staff.

As President-Elect, I devoted my effort to increasing membership, and as your President, I shall do my best to represent you to the public. My prime goal is to increase service to our members and to entice a larger percentage of our members to become active and involved.

The reason for this organization is to help you, its members, to solve problems in order to fulfill the major mission of our profession. I pledge my every effort to the solutions of our collective and individual problems.

It is my good fortune to have been born into a medical family. My father, my brother, and several cousins are doctors. The type of practice conducted by my father was vastly different from the average practice of today. He had a black bag, a large office practice; he made house calls. He was revered by his patients. He was a poor collector. He did not know the words "No," or "I'm too busy." He had very few specific drugs and there was no postgraduate training easily available. He reflected the science of his day, but he was 100 percent physician and he somehow found time to be his patients' part-time lawyer, their part-time minister, and always a friend. He assumed responsibility for the total comfort of those to whom he ministered. All of us know physicians who have developed this type of relationship with patients and probably have experienced this type of relationship ourselves. However, we know that the possibility of this type of close physician-patient relationship continuing into the future is decreasing day by day.

What is happening today is that the practice of medicine and the delivery of health care is changing as society is changing. As a result, our close relationship with our patients is changing. This is all brought about by the explosion of medical and health knowledge in the last 25 years. The age of specialization and the involvement of many professionals in the delivery of care brings to the patient a feeling of de-personalization. We sometimes become so absorbed in the science of the disease that we forget the little things that make the patient comfortable and show that we care. Our patient-physician relationships are sometimes only casual.

We seemingly are solving the major problems in scientific medicine; witness the advances in surgery — transplants, prostheses; and the advances in wonder drugs; witness the antibiotics, cardiac and hypertensive drugs. Advances have been made in genetics and immunology. But are we fulfilling the expectation of our patients and the public? According to most polls today, physicians still have a high rating with patients individually. But collectively, we are criticized because of the cost of health care and the lack of someone to respond readily and economically to the minor illnesses of everyday life. We are being portrayed as the main factor in the increasing cost of health care,

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* P. O. Box 428, Estill, S. C. 29918

even though only 19.8 percent of that total cost is involved in physician fees.

As third parties — government and private — play an increasing role, they tend to dictate where the patient will be treated, even how often the patient will be seen, how many days a patient may be hospitalized, what drugs may be used. All of these further breach the close patient-physician relationship.

Government gives three reasons for its ever-increasing involvement in health matters. They say they want to improve the quality of care, improve accessibility to care and the cost of care. These are laudable goals. The medical profession strongly supports each of them. But they are not mutually conducive to achievement. In the time of double digit inflation, it is impossible to both increase the quality of care and the accessibility to care without increasing the cost of that care. Economists have learned that the economics of health care are different from the economics of industry. In industry, advances in technology increase production and lower cost. In medicine, advances in technology mean better diagnostic procedures and better therapy but they add to cost. In this manner, medicine is like our space program. In landing a man on the moon, NASA learned that to obtain the last two percent of performance necessary to obtain total accuracy cost as much as the first 98 percent of the project. We must support the elimination of waste. We must support all cost reductions that do not result in denial of essential services to patients or that do not interfere with the delivery of quality care. Government will deny payment for services, or place a cap on expenditures. You, the physician and the hospital, must deny the services. It places the profession and the hospital in a "No Win" position.

As a matter of fact, there are attacks being made on the very foundation of our medical profession. We all know about the assault by the Federal Trade Commission on the medical profession's time-honored Code of Ethics. We are told that we no longer can take action to prohibit a physician from advertising his services, regardless of whether or not he is qualified to deliver those services. We are told that we may no longer prohibit individual physicians from associating professionally with practitioners of non-scientific cults and healing methods. We are told that we can no longer demand that all patients should have a right to choose their physician. We are seeing the demand for the licensing of independent paramedical practitioners who may practice their profession independent of physicians and in accordance with their own rules of practice. We are seeing the development of practice norms and criteria which many physicians refer to as "cookbook medicine."

Recently, Federal Trade Commission attorneys have recommended a suit against the American Society of Plastic and Reconstructive Surgeons. It charges that plastic surgeons' reliance upon and support of Board Certification as a standard of competence among those who call themselves plastic surgeons have unfairly restrained physicians who are not Board certified. This argument rejects government's interest in quality, with the apparent theory that costs can be lowered if quality standards of medical care can be lowered.

And from Washington we have proposed legislation which would result in the rationing of hospital care. This is the President's Cost Containment Bill. Here we have a single industry that is to be brought under governmental control because its costs are "too high." Persons working in health care know that a large part of the cost in hospitals is due to inflation caused by deficit spending and excessive government regulations, and the requirement by government that hospitals meet all sorts of "Standards."

In describing the kind of despotism in which Democratic societies like ours could be most vulnerable, Alexis De Tocqueville foresaw a government that covers the "whole of life with a network of petty, complicated rules that are both minute and uniform." A situation, he warned, that does not "break the human will so much as it softens, bends and guides it."

There are those in this nation who feel that the Tocqueville vision is rapidly becoming reality. They point to the enormous and proliferated body of laws and government regulations now controlling virtually every aspect of human life and behavior. The hospital and medical care field is no exception. In fact, it seems to me that we are the focal point.

Physicians and other health professionals have observed the development of these new governmental rules. We have criticized many of them and we have attempted to direct many of them into channels which would not be detrimental to patient care. However, we believe that not only our leaders in government, but the American public should pause before we change the entire system and evaluate the positive changes that are occurring daily in the health of our citizens.

In a recent report of HEW entitled, "Health, United States 1978," Secretary Califano wrote in a memorandum to the President the following: "This report shows that we have made considerable progress in improving the health status of Americans. The average life expectancy continues to increase. The overall death rate stands at an historic low level. The infant mortality rate continues to decline. Mortality from ischemic heart disease decreased by 11 percent between 1968 and 1976." These are encouraging and positive facts, but the Secretary continues by stating, "certain serious problems remain — heart disease and cancer continue to be the two leading causes of death, accounting for nearly 60 percent of all deaths. Mortality from cancer of the lung and other respiratory organs rose in 1976 as well as death from chronic respiratory disease." He continued, "Despite evidence of an increased health risk from smoking, 42 percent of the men and 32 percent of women smoke." Here the Secretary has touched on what most health professionals and others are beginning to realize is perhaps the leading cause of health problems in our nation. That is the life style of our society. First, we have said that the two leading causes of death in our country are life-style related diseases. There are certain types of cardiovascular disease and certain types of cancer. The third leading cause is also related to life style. That is, death from accident. Not only motor accidents, but accidents of all types. These three causes of death account for almost 70 percent of all reported deaths in our country. Far down the list of causes, and comprising not more than 20 percent of deaths, come death caused by such things as influenza, diabetes, bronchitis, tuberculosis and other diseases.

If we include with the life-style related causes of death such things as death caused by homicide, suicide, and cirrhosis of the liver, we find that upwards of 80 percent of all deaths could be related to life-style conditions.

The National Infant Mortality rate in 1977 was 14 per 1,000 live births. In South Carolina, this rate was 17.6. Infant mortality rates vary by race. In South Carolina, the rate is 24 per 1,000 in the non-white population, while in the white population it is 14 per 1,000. This is a variance of 10 per thousand. The reasons for these poor statistics are multiple, but foremost among the causes are socio-economic and life-style causes such as teenage pregnancy, failure to seek medical attention, inadequate personal care, and education. Yet many people conclude from our infant mortality statistics that our medical care system and the doctor is at fault, or at least is inadequate.

We believe an understanding of the factors that contribute to this statistic will show that the medical delivery system alone is not able to change these statistics, even if there were a doctor on every street corner available 24 hours a day.

An example of this was reported in the document, *A New Perspective on the Health of Canadians.* The Canadian government supported a program of National Health Insurance and resource allocation that made traditional health services accessible to all of its citizens. However, the health status of Canadians did not significantly improve as a result of these efforts, and it was concluded that factors other than the organized health care system must be considered. Thus, the Canadian government re-directed its national health policy, focusing on a concept which proposes that health status is influenced by human biology, the environment, the life-styles of citizens, as well as the health care delivery system. Approaching health problems from this perspective gives equal emphasis to those factors which constructively promote health and those that attempt to reconstitute health once it has been lost.

And, of course, government has illustrated that its crystal ball is not any more accurate or foresighted than the crystal ball of the rest of society. We need only to look at the recent involvement of the federal government in the training of health professionals to illustrate this point. For the past several years, the federal government has actively pursued the concept of training more physicians. Now they say train less physicians; stop funding certain programs. Medical schools and teaching hospitals are now in a position of having to seek new methods of financing the training programs which were developed at the encouragement of federal government and are now being abandoned by that same government.

Even with an increased number of physicians, few new graduates are moving into rural and inner city ghetto areas. Trying to address this question and the question of maldistribution is difficult. It is a complex problem that defies a simple solution, and it is one in which our Association, along with the Hospital Association, has been involved. Perhaps the answer will lie within each community to find ways to accommodate these newly trained professionals. Perhaps there should be joint community efforts to foster itinerant professionals who act as referral resource. We would encourage the new graduates to enroll with our Physician Recruitment/Referral Program at the South Carolina Medical Association headquarters, and urge that they look into some of the less physician-populated areas of the state to establish their practices.

Tonight we should spend some time discussing the positive goals that medicine believes should be addressed in the future.

The SCMA and the medical profession supports and actively participates in voluntary cost containment efforts. With the South Carolina Hospital Association, state agencies and health insurance carriers, we have formed the South Carolina Voluntary Health Care Cost Containment effort. We will continue, through this mechanism, to exert influence over unnecessary expenditures in the health care field. As for physicians' fees nationally, they have increased less since August of 1977 than the overall consumer price index.

We believe efforts should be made to reduce the cost of health insurance. We would urge that each policy issued in the state have as a part of that policy payment for ambulatory care benefits. This would discourage use of the more expensive health care services, such as required hospitalization to perform minor surgery, hospitalization to pay for certain diagnostic and laboratory procedures. Many policies pay for emergency room-clinic visits, but deny office visit payments for the same condition.

The South Carolina Medicaid Program continues to be a major program providing payment for health care services to welfare recipients. We believe that where tax monies are used to pay for services to the population, they should be used competitively by both the private and public sectors. Cost reimbursements to public agencies or clinics that are already subsidized by other tax monies for services that can be delivered at less cost in the private sector is not a responsible manner of handling the tax dollar. For instance, the Medicare/Medicaid programs reimburse publicly funded rural health and community health clinics at a rate approximately three to four times greater than they reimburse private physicians for comparable services. Making all services commonly available through both the public and private sectors would increase accessibility and probably reduce the unit cost. Many of these issues are under discussion with the Department of Social Services at this time.

We commend the State-wide Residency Training Programs, feeling that these will render a service to the state in encouraging better distribution of physicians. We feel that the residency programs will probably require expansion and that there should be at least one first year resident slot for each graduate. It is going to be expensive, but it little behooves us to graduate a large number of physicians and send them out of state for their terminal training.

We commend the expansion of the Area Health Education Centers so as to cover the entire state. This gives many of the smaller hospitals a chance to upgrade their educational and training programs.

Earlier, we spoke about the casual effect of the life style of our society on the health of society. As a result, we believe that heavy emphasis must immediately be placed on health education of the public. Appropriate agencies and bodies should be gathered together to form a Task Force to research the effectiveness of health education programs and make recommendations for program guidelines and development. There is in existence a model law for consideration by state legislatures to provide for the establishment of a comprehensive health education program in elementary and secondary schools, grades one through twelve.

Some of our educational institutions have already moved in this direction, and I would personally like to commend the University of South Carolina for being one of the leaders in higher education in offering a degree in Health Education.

I would urge that the Auxiliary to the SCMA consider the promotion of health education in public and private elementary and secondary schools as one of their programs for the coming year.

Having stated some of our possible goals for the near future, I would like to point out to physicians that these goals cannot be achieved by medicine alone. Not only will it require the participation of other health related professionals and organizations, but it will require participation by government at the State and Federal level. In a complex society such as we have developed, we clearly recognize that government must be participant in any meaningful progress which is achieved.

At the same time we are urging government participation, we must caution physicians that while we may act in partnership with government, we will never achieve the status of equal partners with government. Government agencies are established to implement and administer State and Federal laws. In participating with government in the implementation of these laws, citizen groups such as the medical profession will always take a second seat to the requirements of the law which is being implemented. Contractual agreements with government are, at best, one-sided. They can be changed by simply changing the law, and the contractual agreement is thus changed without your involvement.

While recognizing this, we would urge the representatives of government and agency administrators who are here to continue to include the medical profession in your planning and your implementation process in health matters. We would request that they be specific of the problem in medical and health needs as you see them and the needs of the public. The medical profession is part of the solution to the delivery of quality care, not part of the problem. We would like Agencies to realize that while their purpose is to implement law in the health field, our purpose as physicians is to see that those laws do not have a harmful effect on quality care, or hamper the physician's ability to determine it. We of the profession seem to do best when we are advocates for our patients. If we can both respect each other's goals, the outcome of new programs will be improved. We both seek to improve the health status of the people of South Carolina.

While we are discussing the purpose of government in implementation of laws, perhaps we should discuss for a moment that part of government which makes the law — that is the political process.

We hope that physicians and their spouses in South Carolina have accepted the reality that the political process, the election of candidates for public office and the legislative process of enacting laws, is and will continue to be the most dominant influence on the practice of medicine in the foreseeable future. It is not logical for a physician to believe that he can ignore participating in the political and legislative processes and thereby escape the influence of these processes on his practice and on his life. The political and legislative processes by their very nature are commendable pillars of American society. If politics become involved in actions which are perceived to be unacceptable, it is largely because honest, ethical citizens have allowed individuals who do not subscribe to our codes of honesty and ethics to control the political process.

Many individuals in our society voluntarily contribute time and support to the political process to make it work. It is time that physicians enter into this process fully and completely.

There are holders of public office at each end of the political spectrum, but these individuals only make up a small minority of the public office holders. Some 10 percent of our office holders may be willing to socialize medicine tomorrow, and perhaps another 10 percent would vote to totally exclude government from any involvement in health care. But those in the middle, the 80 percent remaining, will base their decisions on the facts given them, and will support them if they perceive them as being in the public interest. If they do not have appropriate physician input in the political process, their decisions will be made by those who do participate in the process.

Along with participation in our political process, we must also point out the need for unity among physicians in South Carolina. Doctors, by the very nature of their training, find it difficult to simply become a cog in a complex organization such as a medical association. Doctors, also because of the demand on our time in our local hospitals and communities, find it difficult to participate in the activities of our State and National Associations. But I would urge you during the coming year to decide to unite with your colleagues in your county and state medical associations and in the American Medical Association.

Through this type of united action, we will achieve the necessary impact on government agencies as they continue to foster new and radical changes in our medical system.

Who will help us in the process, you may ask? My answer to you is the American people — your patients and mine. The influence that they are willing to exert in our behalf is dependent upon how they perceive the profession as a whole and each of us as individuals. Maybe we should strive to reach a half-way point between the practice of yesterday — my father's day — and the science of today. Be scientific? Sure, but deliver quality care with a humane attitude. Yes — be sympathetic.

Our profession is founded on the concept of service to our fellow man. No other profession has the same opportunity as ours. No other can serve the whole man — mind, body and soul. Each of us, by what we do, either spoken or by our actions, is a positive or negative token of the Great Physician. You of the profession are special people — more is expected of you. And if I am any judge of my peers, you do give more, and I feel that you can be proud of your accomplishments. Be apologetic? No. Just continue to deliver service, always trying to improve. You deserve a better shake than is being given to you in some quarters.

As I close, let me read this "Physician's Prayer," published in an Augusta newspaper, on "Doctors' Day" this year. It would serve us all well:

"Lord, who on earth didst minister
To those who helpless lay,
In pain and weakness, hear me now
As unto Thee I pray.
Give to mine eyes the power to see
The hidden source of ill;
Give to my hand the healing touch
The throb of pain to kill.
Grant that mine ears be swift to hear
The cry of those in pain;
Give to my tongue the words that bring
Comfort and strength again.
Fill Thou my heart with tenderness
My brain with wisdom true.
And when in weariness I sink,
Strengthen Thou me anew.
So in Thy footsteps may I tread,
Strong in Thy strength always.
So may I do Thy blessed word
And praise Thee day by day."

Editorials

DOING GOOD WELL

The story goes that Robert Frost was trying hard to counsel his daughter, about to graduate from college and uncertain about what to do with her life. "Well," said the poet, "it really doesn't matter very much what you actually *do*, as long as you do it *well*." "No daddy," objected the younger Frost, "that's not the point — the point is that whatever you do you should be doing *good*." After a long silence, the elder Frost offered a solution: "It doesn't matter very much what you actually *do*, as long as you do *good well*."

Critics of medical organizations such as ours not infrequently maintain that we are not really concerned with doing *good*, but are more preoccupied with our own self-interest. To argue otherwise, we should begin with a look at our history. A century ago, the president of the South Carolina Medical Association observed the "depressed condition of medicine in South Carolina," with "charlatanism in the cities" and with apothecaries — rather than physicians — rendering "at least one-half of the ambulatory medical practice." Due to the initiative of the SCMA, the state Legislature appointed a Board of Health and in subsequent years our medical association fostered the emergence of both public health and of scientific medicine in our state.[1] We *began* with the intent to do good — this is our heritage.

A careful scrutiny of this year's presidential address by Dr. Harrison Peeples should convince any critic that doing good remains our intent. Commenting on the impact of lifestyle on health and disease, Dr. Peeples proposed that a task force be created to study methods for implementing health education programs. Such a task force would include members of the health professions, consumer groups, hospitals, nursing homes, the Department of Education, the Department of Health and Environmental Control, and other groups. Recognizing that modification of life styles by individual physicians for individual patients, one on one, is both expensive and slow, Dr. Peeples proposed a *comprehensive health education program* for elementary and secondary schools. A century ago, an SCMA president proposed a Board of Health; now our president, again recognizing a public need, has proposed another measure for the public *good*.

But how to do it *well*? It cannot be denied that the role of our association has changed dramatically during this century. Government now assumes most of the public health functions once provided by the South Carolina Medical Association. Specialty societies and medical school faculties now assume most of the scientific functions which the medical association once provided. Here again, the answer appears among Dr. Peeples' messages. His essay, "In unity there is strength," in the May issue of *The Journal* makes the key point that the SCMA can be the one *umbrella organization* which can give our profession the power to bring about changes and programs which are in the public interest.

These thoughts — the need for improved health education and the need for unity in our profession — are of course not novel, and one might ask whether Dr. Peeples' emphasis is appropriate for the medical profession in 1979. Should we leave trends in health education for others? Is the notion of *unity* in the medical profession obsolete? To answer such questions, it is sometimes useful to listen to the philosophers, who sometimes have greater insight into our lives than we would like to admit.

A recently published essay entitled "A framework for responsible medicine" may be relevant.[2] The author begins with the statement that "the construction of an ethic for the profession of medicine in a radically changed society is a formidable task entailing the clarification of the dimensions of the profession and the extraction of an applicable ethic from a bewildering mass of historical and philosophical materials." After struggling through this and similarly abstruse passages, I came away with the following conclusions:

1. Medicine is "no longer a private club." The public increasingly pays for our services and therefore deserves an accounting from us on our "total performance in delivering health care."
2. There is an obvious need for health education: "We do not as a nation understand or value health. As a result we waste vast amounts of the nation's wealth in repairing our bodies. . . ."
3. It is the medical profession's *obligation* to find an answer to the lack of a "societal commitment to health."
4. The medical profession if "*unified in a single organizational unit* could undertake such an educational project" (italics mine).
5. In the context of such an educational project, the role of the medical profession would be "not so much to do this themselves but to support and to recommend to the public those health professionals whose primary function is already educational."

It seemed, then, that these conclusions were virtually identical to those offered by Dr. Peeples. We have not only the opportunity to do good; we have a *mandate* to provide some solutions. Only by unifying in strong umbrella organizations — such as the SCMA — can we do it *well.*

— CSB

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2. Newton L: A framework for responsible medicine. Journal of Medicine and Philosophy 4: 57-69, 1979.

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President's Page

PRIORITIES

If our organization is to avoid being overcome by the everyday maze of minutiae or to avoid being like a ship without a rudder, we must set our course by developing our theme and establishing priorities for the year. Our theme, "Better Health Through Health Education," has been previously discussed (last month's President's Page and in my Inaugural Address published in this issue). We have had offers of assistance in promoting this concept from several sources. We shall pursue the creation of the study Task Force.

I propose the following priorities and request that you send us your recommendations for possible inclusion in the list when it is finalized:

1. **COUNTY AND DISTRICT MEDICAL SOCIETY VISITATION:** I would like to initiate once again the practice of SCMA Officers, Councilors, and staff representatives visiting all county and district societies and presenting programs. We want an opportunity to meet and talk with the membership at home. This will give you a chance to tell us what you expect of the Association. Communication? Yes, that's the name of the game.
2. **SCMA DUES-PAYING MEMBERSHIP:** I would like to recommend that the SCMA for the coming twelve months set a goal of increasing its dues-paying memberships by 20 percent. Since we have a fixed overhead for service to our present membership, about 100 percent of the extra revenues generated from new members could be used for more and better services to you. I would like to ask that each county society and each specialty society establish a committee to increase their membership in SCMA by that 20 percent.
3. **PROFESSIONAL LIABILITY:** I recommend that during the next twelve months we develop with in the South Carolina Medical Association a firm position regarding a permanent market for the physicians' professional liability insurance. We should, once and for all, develop answers to such questions as "Should the JUA be permanent? Should it be merged with the Patients' Compensation Fund? Should the SCMA seek to administer these programs, and what will be our position regarding the continuation of the CNA program?"
4. **COMMISSION/COMMITTEE STRUCTURE:** I recommend that we implement the new Commission/Committee structure as outlined in our Constitution and Bylaws and, at the same time, implement a corresponding staff component to each Commission and Committee. I would like each Commission to study, in its broadest sense, the subject encountered in its title, and bring to Council recommendations for activity by the Association; that is, public health issues, socioeconomic issues, scientific activities and medical education, public and professional relations. This would encourage input from a larger percentage of our membership.
5. **THE SCMA ANNUAL MEETING:** I recommend that we completely restudy the purpose and function of our Annual Meeting and determine whether it shall be only a business meeting of the House of Delegates or whether it should be developed as a meeting function to offer activities which will attract all of our South Carolina membership. Should we develop a separate scientific and CME conclave that would involve all specialties? How best can the Annual Meeting meet the needs of the membership?
6. **RELATIONSHIPS WITH NON-M.D. HEALTH PROFESSIONALS:** We have had a major start in defining the relationship of M.D.s with the nursing profession and with their educational processes. We have yet to establish clear channels to implementing these goals, and we have not yet addressed the long-range relationships of M.D.s to other health professionals such as midwives, physicians' assistants and reimbursement to these professionals. I would like to assign these goals and objectives to the Ad Hoc Committee on Non-M.D. Health Professionals.

I intend to ask Council to establish a full list of our priorities. These will serve as our rudder to bring to fruition our goals of better service to you.

What do you think should be added?

Harrison L. Peeples, M.D., *President*

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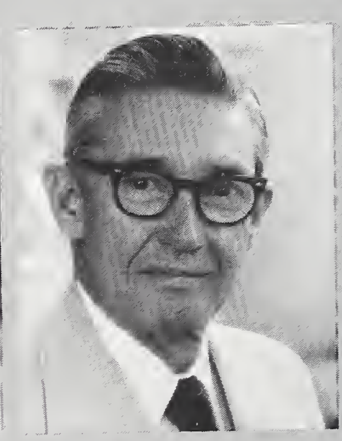

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CONVENTION HIGHLIGHTS

Dr. John Hawk addresses House of Delegates. In background is Dr. Halsted Stone.

Three past State Presidents are Mrs. Lucious M. Cline, Jr., Mrs. Leonard Douglas, and Mrs. Rufus H. Cain, Jr. In foreground is Mrs. Manuel A. Bergnes, President of American Medical Association Auxiliary.

1979-80 Officers of the SCMA Auxiliary.
President-Elect — Mrs. James F. Adams, Jr.
Vice-President — Mrs. Arthur M. LaBruce
Treasurer — Mrs. R. Maxie McCoy

Guest Speaker, Marguerite E. Tracy, author of "Doctors Die, Too." She spoke on "Finances for Women" at the breakfast hosted by Southern Bank and Trust Company.

Outgoing President, Mrs. Alton G. Brown presides at Executive Board Meeting. Looking on is Mrs. William P. Turner, Jr., Parliamentarian and Timekeeper.

Dr. Alton G. Brown presents wife, Nellie Claire with the Past President's Pin.

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DIAGNOSTIC ULTRASOUND: ITS PLACE IN THE EVALUATION OF RENAL DISEASE*

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Renal diseases were among the earliest investigated by diagnostic ultrasound[5] and with recent advances in grey scale equipment, the indications for ultrasound and the diagnostic information obtainable has increased dramatically.[10] A growing number of Diagnostic Radiology Departments are now providing ultrasound services making it available for the evaluation of most patients. The purpose of this paper is to report the experience with renal ultrasound at the Medical University of South Carolina and to review the indications for, and the usefulness of this procedure.

TECHNIQUE OF RENAL ULTRASOUND

The examination is performed with the patient prone. An acoustic coupling agent, mineral oil or gel is applied to the skin of the back and longitudinal scans at 1 cm intervals, parallel to the vertebral column through both kidneys are obtained (Fig. 1A). Transverse scans at right angle to the vertebral column at 1 cm intervals are then made (Fig. 1B). The right kidney may be viewed through the liver with the patient supine (Fig. 5). Ultrasound images are recorded on either radiographic or polaroid film.

The procedure takes from 20 to 40 minutes to complete and aside from the need for lying prone, the examination is free of patient discomfort. As with all diagnostic ultrasound examinations, no ionizing radiation is employed and there is no known risk.

NORMAL RENAL ULTRASOUND

Normal kidneys are visible on ultrasound in longtudinal sections (Fig. 1A) as eliptical organs with sharp margins at the perirenal fat. The cortex produces fine low level echoes, less dense in intensity than the adjacent liver or perinephric fat. The medulla is almost completely lucent and arcuate vessels may be imaged allowing measurements of cortical thickness. Peripelvic fat, caliceal system and vascular structures combine to produce dense central echoes at the renal hilum.[10]

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PATIENT MATERIAL AND RESULTS

From June through December 1978, 222 patients underwent 255 ultrasound examinations at the Medical University of South Carolina.

A large number of patients were examined because of recently diagnosed renal failure to detect renal size and/or bilateral obstruction (118) or the presence of adult polycystic disease (10). Unilateral non-function (15), and renal masses (55) (on intravenous pyelogram) were frequently evaluated to detect hydronephrosis and to differentiate solid from cystic masses. Interventional procedures on the kidney with ultrasound guidance included localization for percutaneous renal biopsy (18), cyst aspiration (8), and percutaneous nephrostomy placement (6). Other indications included evaluation of renal transplants for obstruction or perinephric fluid (6), localization of kidneys for radiation therapy (1) and the evaluation of renal and/or perirenal infection (14). When conventional x-ray procedures, such as IVP, were contraindicated due to pregnancy (3) or contrast material allergy (1) ultrasound was occasionally performed.

DISCUSSION

MASSES, CYST VERSUS SOLID

One of the most important uses of renal ultrasound is in the evaluation of renal mass lesions.[5, 8] Particularly in older patients, simple renal cysts are extremely common[2] and as a result excretory urograms obtained for other reasons such as benign prostatic hypertrophy or urinary tract infection often disclose asymptomatic renal masses. The radiographic appearance alone is often insufficient to confidently exclude renal cell carcinoma and prior to ultrasound, renal angiography and/or exploratory surgery were often necessary.

Ultrasound is able to distinguish between cystic, fluid-filled masses such as simple renal cysts and complex or solid ones by the quality of the reflected sound waves. A cyst without internal structures will not produce internal echoes even at high sound intensities, and very little attenuation of the sound beam will occur between the front and back walls giving a strong posterior wall echo.[5] (Fig. 2A) Solid masses will almost always have numerous tissue interfaces within them which will cause internal echoes often making the mass more echo dense than the surrounding normal kidney. (Fig. 3A) Attenuation of the sound beam will weaken the intensity of the posterior surface even in relatively lucent solid masses.[1]

When a mass demonstrates all of the qualities of a cyst, strong through transmission of sound and echo-lucency, even at high sound intensity, a diagnosis of cyst is virtually assured. If clinical suspicion of significant pathology persists, percutaneous needle aspiration of the cyst guided either by ultrasound or fluoroscopy and examination of the cyst fluid both cytologically and chemically for signs of malignancy can be performed.[2] (Fig. 2B)

If ultrasound reveals internal echoes within the mass or a weak posterior wall due to sound attenuation, further evaluation including angiography is warranted to exclude malignancy.[8] (Fig. 3B)

The majority of renal masses prove to be simple cysts and most have typical ultrasound appearances allowing the patient to be spared further uncomfortable and potentially hazardous investigations.

RENAL FAILURE

When a patient presents to a physician and is discovered to have renal failure (elevated BUN, creatinine) it becomes important to differentiate between chronic renal failure and acute failure such as might occur from obstruction. (Fig. 4) Ultrasound is ideally suited for this differentiation. Excretory urography which depends upon renal function often will not allow adequate visualization for a diagnosis to be made. Ultrasound will allow accurate measurement of renal size, detection of caliceal dilatation and judgement of cortical thickness.[10, 11] Occasionally a patient with polycystic renal disease will present with undiagnosed renal failure. The ultrasound images are characteristic with large kidneys filled with numerous large and small cysts. (Fig. 5) Infrequently hepatic and pancreatic cysts will be visualized as well.[6]

RENAL LOCALIZATION

With more advances in the management of renal diseases, accurate histologic diagnosis has become more important. Because of its ease and speed of performance, percutaneous needle biopsy of the kidney is frequently the procedure of choice. Ultrasound is a useful aid in renal biopsy. The lowest and lateral portion of the kidney can be rapidly and accurately localized even without significant renal function, and the depth of the kidneys from the posterior skin surface accurately measured.[7] Renal ultrasound can be performed immediately prior to renal biopsy, and because the procedure itself is quite rapid, the biopsy can be performed in the same room. Localization by ultrasound is particularly useful in patients with small or misplaced kidneys.

NON-FUNCTIONING KIDNEYS

When a single functioning kidney is visible on excretory urogram, the differential possibilities include absence or severe atrophy of the contralateral kidney, vascular obstruction, hydronephrosis and neoplasm. Again ultrasound is very useful.[9] If the non-functioning kidney is small, either atrophy or severe vascular insult is most likely. When obstruction is present the degree and often the site of blockage can be outlined, and the amount of remaining renal parenchyma can be judged. A kidney replaced by a solid mass suggests tumor invasion. In a child, multicystic dysplastic kidney and Wilms tumor also enter the differential; multicystic dysplastic kidney, however, may be difficult to differentiate from a high grade urinary obstruction and Wilms tumor is usually indistinguishable from other solid neoplasms.[13]

MISCELLANEOUS DISEASES

A variety of other less frequently encountered clinical problems and diseases have been studied by ultrasound. Relatives of patients with polycystic renal disease may wish to know if they are affected and ultrasound avoids ionizing radiation which may be important in the younger patients. If renal, hepatic or pancreatic cystic disease is documented, the diagnosis can be made. Unfortunately cysts in this disease grow with time and a negative ultrasound examination does not exclude early polycystic disease.[6]

In the very young and in pregnant females, the desire to avoid irradiation if possible is always present. The usual diseases being investigated are nephrolithiasis and infection and unfortunately a negative ultrasound examination does not exclude stones or renal infection and excretory urography still generally needs to be performed.[12]

Nephrolithiasis, particularly with lucent stones, has been investigated by ultrasound and stones may be visible even when difficult to detect on excretory urograms, but the latter still remains the examination of choice in almost all patients.[4]

Ultrasound has been shown to be very useful in the evaluation of kidneys following renal transplantation. Perirenal collections of fluid and lymph, changes in renal size and the presence of renal edema in rejection can all be visualized by ultrasound.[3]

Perirenal fluid collections, particularly perirenal infection can be easily localized by ultrasound as fluid collections in the perirenal space and aspirated when appropriate for culture.[12]

In conclusion, diagnostic ultrasound has become an accepted imaging modality in the evaluation of renal pathology. It allows painless, rapid and inexpensive evaluation of the morphology of the kidneys without relying on either renal function or ionizing radiation. As technical advances in equipment design continue to occur, the quality of ultrasound images and the information provided should continue to improve. ☐

Acknowledgement:
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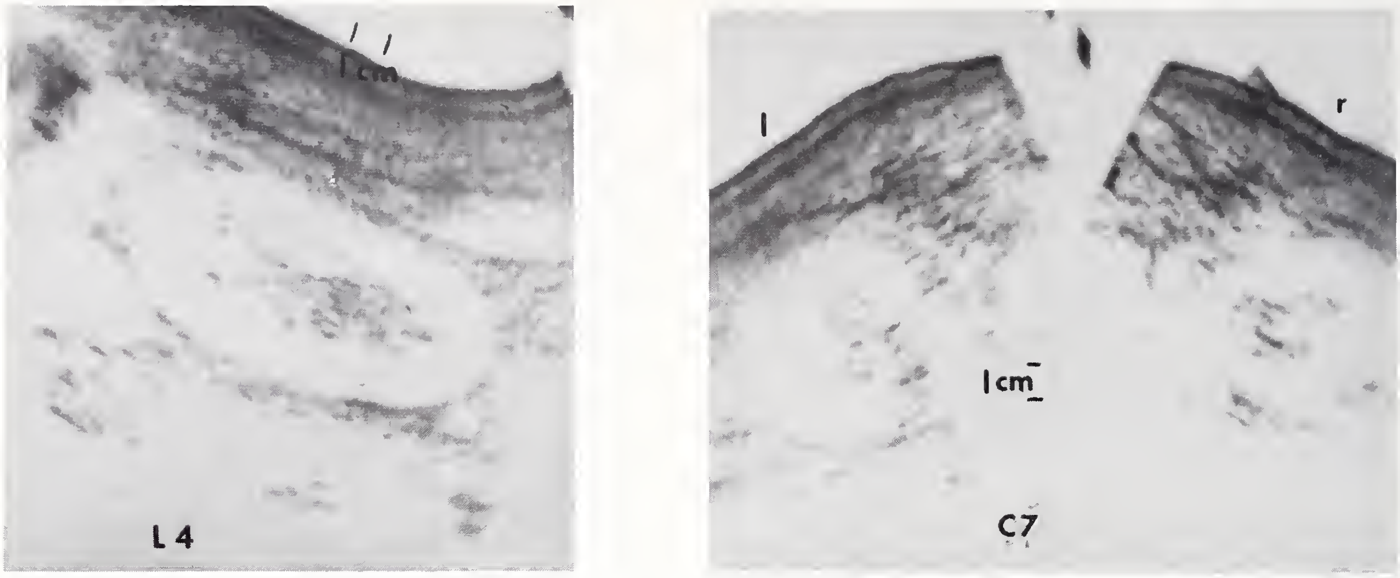

FIGURE 1 — Normal Renal Ultrasound

A. Longitudinal scan 4 cm to the left of midline shows a normal left kidney with an eliptical shape. Renal parenchyma is lucent relative to the dense central echoes formed by calyces, fat and blood vessels.

B. Transverse scan 7 cm above the iliac crest viewed from below demonstrates normal R and L kidneys in cross section.

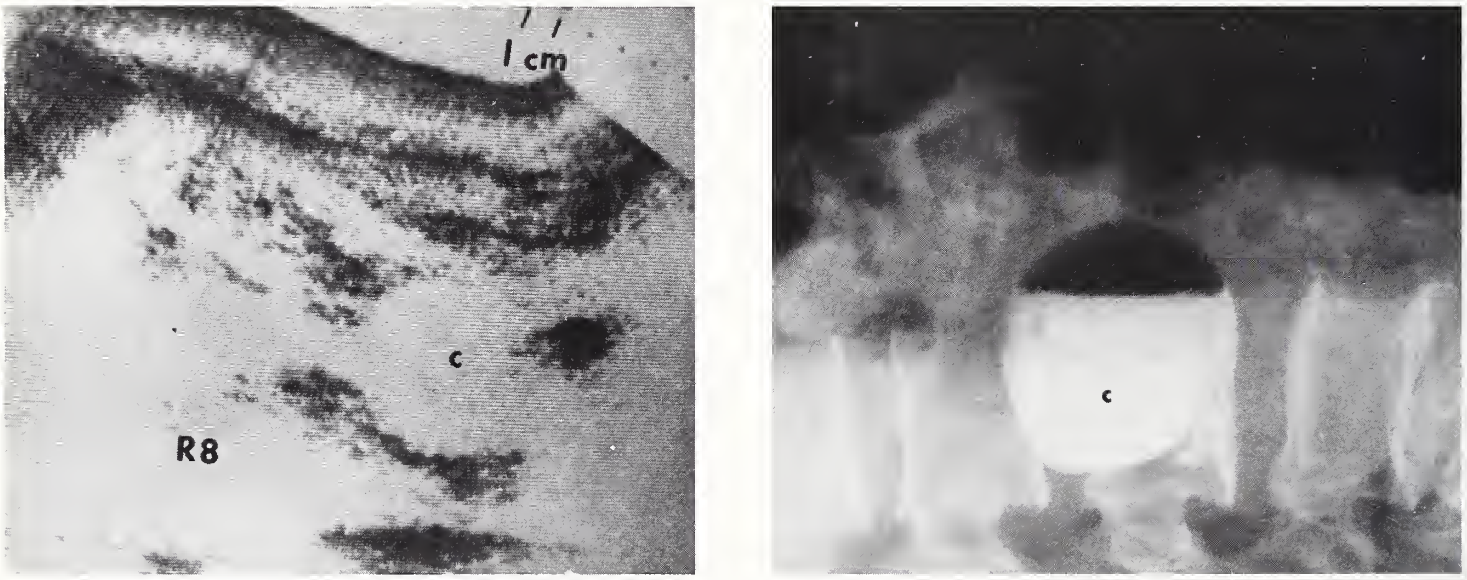

FIGURE 2 — Simple Renal Cyst

This 63 year old male was discovered to have a mass in the lower pole of the R kidney on an IVP performed for evaluation of benign prostatic hypertrophy.

A. Longitudinal ultrasound scan 8 cm to the R of the midline demonstrates a somewhat atrophic R kidney with a 4 cm sonolucent mass in its lower pole. (Note the strong ventral cyst wall echoes.)

B. Percutaneous cyst aspiration with ultrasound guidance yielded clear fluid with normal chemical and cytological analysis. Water soluble opaque material and air injected at the time of aspiration outlined the smooth cyst walls.

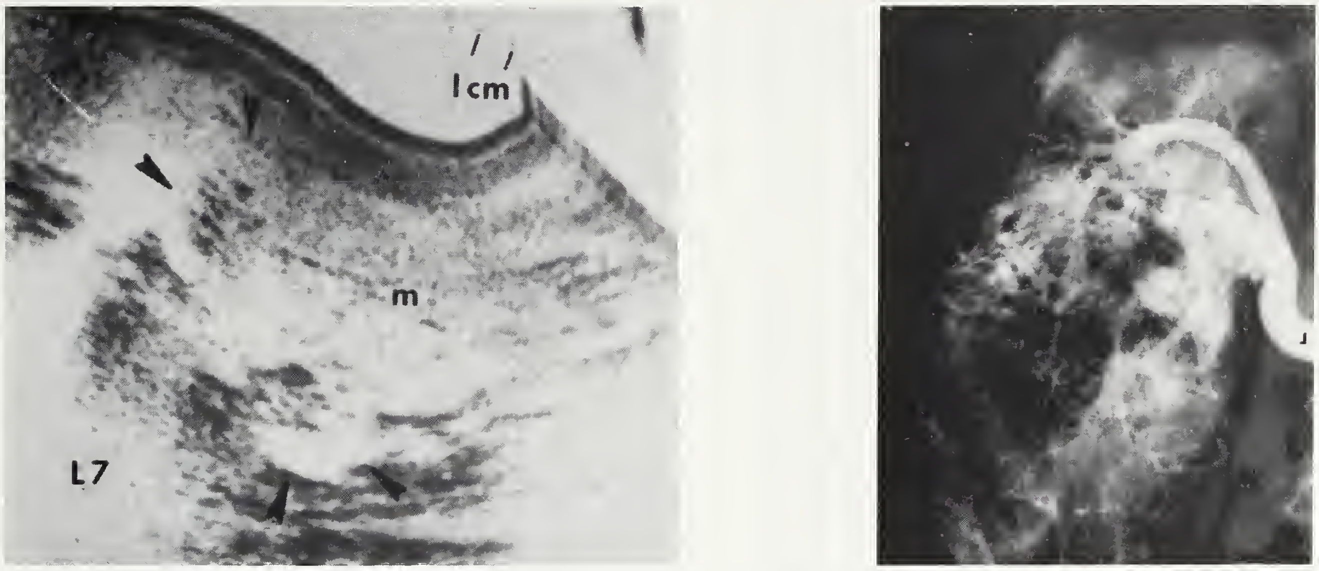

FIGURE 3 — Solid Renal Mass

This 58 year old female with microscopic hematuria was found to have a L renal mass on IVP.

A. Longitudinal ultrasound scan 7 cm to the left of midline demonstrates a 5 cm mass (M) with dense echo production in the mid portion of the L kidney. The upper and lower poles of the kidney (arrows) are separated and the lower pole pushed ventrally.

B. Selective L renal arteriogram confirms the presense of a solid tumor which is very hypervascular and proved to be a renal cell carcinoma at nephrectomy.

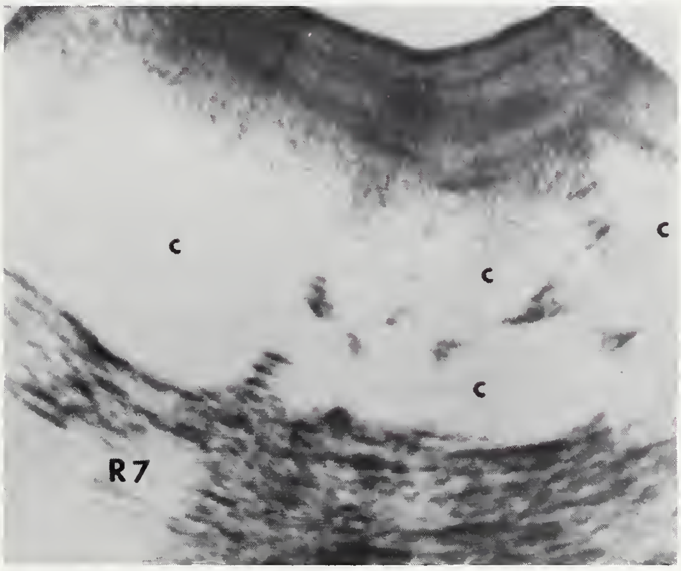

FIGURE 4 — Non Functioning Kidney
This 43 year old female with carcinoma of the cervix was found to have a non-functioning R kidney on IVP.
Longitudinal ultrasound scan 7 cm to the R of midline demonstrates large sonolucent cystic spaces (C) corresponding to massively hydronephrotic calyces.
The distal R ureter was found to be obstructed by metastatic tumor.

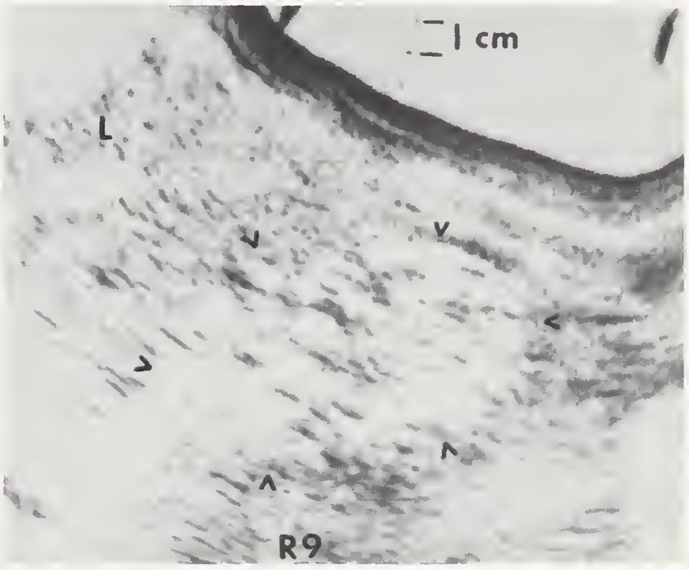

FIGURE 5 — Polycystic Renal Disease
This 41 year old male presented with uremia. There was a family history of renal disease.
Longitudinal ultrasound scan with the patient supine 9 cm to the R of midline demonstrates an enlarged R kidney (outlined by arrows) filled with cysts up to 1 cm in diameter. The left kidney was similar in appearance.

A COMPARISON OF FAMILY PRACTICE RESIDENT ASSESSMENT EXAMINATIONS

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One of the most important issues in the highly complex area of health care is the problem of assessment and assurance of the quality of health care. Brook states "techniques for measuring quality of care are still in their infancy."[1] "Efforts to evaluate the quality of health care are becoming more explicit and more intensive."[2] In the continuing effort to achieve quality health care, a primary focal point must be the physician. As a major figure in the health care team, he has the primary responsibility for delivering quality care. Thus, if the quality of the physician's performance is enhanced, the level of medical care should improve.[3]

The task of improving the quality of physician care is being addressed on many levels: premedical school qualifications, curriculum changes and instructional methods in medical schools, and increasing evaluations of post graduate training programs by accrediting agencies. The increasing requirements are indicative of these efforts. The concern of the practicing physician for quality health care is expressed through participation in self-assessment programs, time spent away from income producing practice in continuing health education programs and in their concern for developing performance evaluation criteria.

The purpose of this study was to examine one dimension of those factors which contribute to quality health care, the evaluation of family practice residents by means of direct testing. Literature on the evaluation of family practice residents revealed 190 approved family medicine residency programs which de Shazo and Davis divided into three categories:[4]

Category 1 — Minimal evaluation in the program. Usually observation of residents with the completion of a simple rating form. Characterized by little or no direct testing and seldom any effort to gather any baseline data.

Category 2 — Some logical method of program evaluation. Programs usually characterized by the use of some direct testing of residents through the use of standard self-assessment examinations. Oral examinations are frequently employed with periodic conferences for progress evaluation.

Category 3 — Employment of comprehensive evaluation mechanisms with pre-assessment of clinical skills of each resident. Frequent progress conferences using data generated by various evaluative techniques were used. A wide variety of evaluation techniques are utilized: Oral examinations, peer review, chart audits, videotape evaluation sessions, patient management reviews, and patient satisfaction surveys.

The vast array of evaluation methodologies utilized by the various family practice residency programs is beyond the scope of this study. Therefore, this study was limited to a comparison of the resident evaluation tests utilized by the Family Practice Residency at Dwight David Eisenhower Army Medical Center, The Core Content Review of Family Medicine[5] and the Individual Resident Profile[6] with the In-Service Resident Evaluation[7] developed by the Division of Evaluation, Department of Family Practice, Medical University of South Carolina.

METHODOLOGY

The study was divided into two phases. Phase I consisted of attendance by the junior author at a two and one-half day seminar on In-Service Resident Evaluation conducted by the Division of Evaluation, Department of Family Practice,

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Medical University of South Carolina, Charleston, South Carolina. Phase II encompassed the review of literature and study of the two examinations utilized by the Department of Family Practice, Dwight David Eisenhower Army Medical Center, Fort Gordon, Georgia in the evaluation of residents.

A comparison of the three resident examinations was then made based upon the following factors:

a. Method of Administration and resources required.
b. Examination content.
c. Domains evaluated by the examination: Cognitive, affective, psychomotor.
d. Data obtained from the examination.
e. Cost.

RESULTS

A. Method of Administration

In the comparison of methods of administration, each examination was found to contain a common element: The use of multiple choice type questions/answers. The Core Content Review and the Individual Resident Profile consisted solely of multiple choice questions. Therefore, the method of administration for the Core Content Review and the Individual Resident Profile as well as the multiple choice section of the In-Service Resident Evaluation were all administered by the traditional classroom/pencil/multiple choice test/proctor method. Time varied according to the number of questions the resident was expected to answer all questions. The Core Content Review may be administered to one or more residents. The Individual Resident Profile, by selecting the examination based upon the residents practice profile, is tailored to individual administration. The In-Service Resident Evaluation, by reason of the elaborate logistic and personnel requirements, is better for testing all residents in a program during the same period.

While completion of the multiple choice questions terminates the examination for the Core Content Review and Individual Resident Profile, it is only the initial phase for the In-Service Resident Evaluation. In this evaluation, a second classroom session is devoted to the patient management problem section of the evaluation. This requires an additional four hours of classroom work solving three lengthy patient management problems.

At this point the similarity in administration ends. As stated previously, the Core Content Review and the Individual Resident Profile are completed with the multiple choice questions; however, the administration of the In-Service Evaluation is just beginning. The In-Service Evaluation still has three sections to be completed; the office oral, the physical oral, and the formal oral, each with attendant problems in administration.

The office oral requires:

1. Equipment and space — an office equipped with a one way glass wall, closed circuit TV camera, a room for the examiners, sufficient space for the resident, patient and three examiners.

2. Personnel — a role playing actor (patient), examiners: two physicians, a behavioral scientist, and a clerical/timekeeper assistant.

3. Exacting time schedules in order that the residents being tested, the examiners and actors all function smoothly throughout the test period.

The physical oral requires:

1. Equipment and space — an examining room with attendant equipment for a physical examination with closed circuit TV camera or one way glass wall plus a room for the examiners/or an examining room sufficiently large for the resident patient and two/three examiners. A patient room where patients may wait between examinations.

2. Personnel — a patient with multiple pathologies who will submit to repeated examinations by the residents. Two/three physician/examiners. A clerical/timekeeper assistant.

The formal oral requires:

1. Equipment and space: an office with desk and chairs for the resident and two examiners.

2. Personnel — two physicians/examiners and one clerical/timekeeper assistant.

These three sections of the In-Service Resident Evaluation require one or two additional testing days dependent upon the number of residents to be evaluated and the number of examining teams to be utilized.

B. Examination Content

Comparison of content reveals great variation between the examinations. The Core Content Review is comprised of four sub-tests covering questions on medicine, pediatrics, psychiatry, surgery and obstetrics/gynecology. Questions are drawn from the bank of questions of the Core Content Review, a self-assessment multiple-choice examination developed by the Connecticut and Ohio Academies of Family Physicians.[8]

The Individual Resident Profile consists of multiple choice questions specifically tailored to the resident being examined. The resident dictates on tape certain information about every patient contact for four to five days. These data collection days may be consecutive or may be spread over a month-long period. The dictated information is transcribed and coded into the IRDA. From this a computerized profile is generated. The test composition is based on the profile so that it concentrates on patient problems occurring with greatest frequency in the resident's training.

The In-Service Resident Evaluation utilizes two multiple choice tests. One is the National Board of Medical Examiners' Special Test in Medicine. The second test is the Departmental Multiple Choice Paper prepared by the Department of Family Practice, Medical University of South Carolina. It consists of ninety questions on Behavioral Science, Cardiology, EENT, Endocrinology & Nutrition, Gastroenterology, Geriatrics, Hematology, OB/GYN, Pediatrics, Pulmonary, Rheumatology and Urology. The multiple choice tests are followed by three written patient management problems. These are prepared by faculty in the Department of Family Practice. The resident then has three observed performance tests: The office oral, whereby an actor portrays a patient with a staged problem and the examiners observe and grade the resident's performance; the physical oral, in which a real patient is given a physical examination by the resident. He is then graded on the completeness of the examination and the reporting on known pathologies of the patient. The third performance test is the formal oral in which an examiner provides information on a patient problem and the resident responds with his management of the case. An examiner also observes the resident and the resident is graded on his investigation, diagnosis and management. Each of the three orals lasts one hour with thirty minutes devoted to the actual performance and thirty minutes provided for critique, immediate feedback and reteaching by the examiners.

C. Domains evaluated by the examination

Examination of the three testing devices confirms that all three test the cognitive domain through the use of multiple choice questions on various medical areas. The In-Service Resident Evaluation tests the cognitive domain throughout the entire six subsections of the evaluation. It also tests the psychomotor domain in the physical oral and the affective domain in the office oral. This is the reason for requiring a behavioral scientist as one of the examiners. The role playing patient also acts as an examiner for that portion of the evaluation.

D. Data obtained from the examination

Examination results for the Core Content Review are reported as Minimum Pass Level, Mean, and Standard Deviation for all residents, then for each year of the program, first, second and third. Scores as reported may be used to assess strengths and weaknesses of individual residents and total programs. Scores may be compared with other programs or national means.

The Individual Resident Profile, by the method of examination question selection, furnishes data on strengths and weaknesses of the individual resident based upon his current patient group. As each resident tested does not answer the same questions, a comparison by mean, Minimum Pass Level or Standard Deviation is not possible. However, the answers are scored and areas scoring below normal levels are suggested for additional study. A summary of the results of all residents can provide a frequency breakdown of the patient problems encountered in the clinics. This can be an aid in planning and a means of identifying group needs for continued education.

The In-Service Resident Evaluation reports the range, mean and individual resident scores for each segment of the examination. The patient management problems are also reported by the number of errors made, the number of errors of commission, and the number of errors of omission for each problem. As the office oral, physical oral and formal oral are subjectively graded by the examiners, the ratings are reported for each resident by examiner with the averages of examiners' ratings. Averages are also reported for first, second and third year residents.

Results are reported grouped by cognitive skills, psychomotor skills, attitudinal skills and overall performance for each resident. Program directors receive comparisons of scores for their residents and other programs participating in the evaluation programs for that year.

E. Cost

Costs of the three examinations vary from $20.00 per resident for the Core Content Review, through $45.00 per resident for the In-Service Resident Evaluation, to $70.00 per resident for the Individual Resident Profile. There are additional expenses in administering the In-Service Resident Evaluation. Resident programs participating in this evaluation program for the first time must pay the costs of a member of the Evaluation Division, Department of Family Practice, Medical University of South Carolina, to be on site and direct the administration of the examination. Programs are also encouraged to send a team consisting of a physician, behavior scientist and administrator to attend an In-Service Resident Evaluation seminar prior to administering the examination. Depending upon distance from the Medical University of South Carolina, these requirements can add considerably to the cost of a testing program.

DISCUSSION

"Evaluation is one of the most widely discussed, but little used phenomena in today's medical education system. Verbal statements about evaluation and accountability are abundant, but genuine evaluations of education programs are rare."[9] Resident assessment examinations have been in use for over a decade. First designed to assist residents in preparation for board examinations, they have gradually developed into examinations designed to aid individual residents and residency programs in assessment of strengths and weaknesses.[10] A prerequisite to a successful personalized program is knowledge about the resident. Without knowledge of competencies and future goals of the resident, relevant advising becomes impossible.[11]

In light of the above statements each of the three examinations serves a purpose in resident evaluation. Each has limitations, i.e., the Core Content Review tests only the cognitive domain using standardized questions; the Individual Resident Profile is best utilized for determining deficiencies of individual residents rather than all residents in a program, and the In-Service Resident Evaluation is not feasible for an individual resident because of the resources required for administration, but is excellent for all residents in a program. Nevertheless, each examination should have but a single common purpose, to enhance learning. Each of the three will accomplish this to some degree; however, the Core Content Review and Individual Resident Profile serve only to point out the presence or absence of cognitive skills. The ability to deliver quality health care cannot be adequately assessed by the results of an objective multiple choice test. Not only knowledge, but attitudes and practice must be observed to effectively evaluate. Observed performance is the key to reliable evaluation. All too often the individual achieving high scores on objective, multiple choice or other examinations requiring the recall of material falls to the bottom of the class when confronted with situations requiring judgment, decisions and performance. The degree of difficulty can only be increased when the situation requiring judgment, decision or performance is known to be observed and rated. This type examination more nearly simulates the environment and requirements of the working physician in a hospital or clinic. He is confronted with real patients, with varying pathologies and must make judgments and decisions under the observation of nurses, ancillary personnel and fellow physicians. He/she is being examined by every patient, nurse, or peer in the cognitive, affective and psychomotor realm, and although ratings may not be expressed, nevertheless, he/she is undergoing evaluation.

These examinations do not lend themselves to the normal means of comparison, validity, and reliability coefficients, but then neither does the practice of medicine. Attitude, patient/physician interaction and many other variables enter into that elusive term, quality care, which itself has not been successfully defined.

CONCLUSION

It would, therefore, appear that the means of examination which most nearly duplicates the actual practice of medicine would be the most valuable assessment of the resident. This is not to say there is no place for examination tools like the Core Content Review or Individual Resident

Profile. These or similar examinations may well have a place in a comprehensive evaluation program, but the In-Service Resident Evaluation most nearly approximates that which should be examined, the knowledge, judgment decisions and performance required in the practice of medicine. ☐

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DOES MCAT PREDICT CLINICAL PERFORMANCE?

DARLENE L. SHAW, Ph.D.*
PATRICIA M. RANDELS, M.D.*
PAUL ELSON, B.A.*

The Medical College Admission Test (MCAT), introduced in 1946, was designed to assess aptitude in basic sciences. While the MCAT has been highly useful in predicting success in the basic science years, there is little evidence that it can predict good performance in the clinical years or in clinical practice. Nor is there evidence that the MCAT is associated with humanistic attributes.

Two studies[1, 2] have followed medical students through the internship year in efforts to determine the predictive utility of the MCAT. Korman and his associates[1] collected a variety of preadmission personality measures, ratings on pre-admission interviews and biographical data as well as MCAT scores and premedical grade point averages. Performance measures included grades in medical school, "physicianship" ratings by clinical and preclinical faculty, and internship ratings by supervisors at the end of the internship year. From a factor analysis they derived four factors with significant corelations with other variables. These factors were labeled: (1) Grades in Clinical Medicine, (2) Internship Success, (3) Scientist Potential and (4) Humanism. Grades in Clinical Medicine correlated well with MCAT scores. Internship Success seemed to be almost exclusively determined by sociability. Scientist Potential was significantly correlated with a number of items including MCAT scores and premedical grade point averages. The Scientist Potential factor was high in students who had been identified in preadmission interviews as individuals who would do well in research but not as general practitioners. Personality measures on students with high Scientist Potential revealed individuals with a preoccupation with achievement, who did not defer to or particularly need others, and who tended to be reflective. Humanism was significantly negatively correlated with "orientation to prestige and prominence" on the faculty interview ratings. MCAT scores and premedical grades were not related to Humanism.

Best and his associates[2] used biographical data (age, sex, type of premedical college, major subject, etc.), premedical grades, MCAT scores, and ratings of letters of recommendation as predictors of performance on: (1) comprehensive examinations taken at the end of each year of medical school, (2) patient management problems, and (3) clinical clerkship ratings. They found that premedical grade point averages and MCAT scores were significantly correlated with performance on multiple choice, comprehensive examinations but not with performance on patient management problems or clinical clerkship ratings.

In addition to studying the predictive utility of the MCAT, some investigators have commented on the personality characteristics associated with high MCAT scores. Gough[3] reported that students with the highest MCAT scores were described as being narrower in interests, less adaptable, less articulate and less comfortable in interpersonal relationships than their lower scoring peers. His study also revealed that MCAT scores were "almost completely unrelated" to faculty ratings of clinical competence.

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Korman and his associates[1] point out that MCAT scores and premedical grades systematically select "smart, achievement-oriented, rather aloof individuals who know how to get good grades." However, these selection tools do not necessarily identify individuals with strong humanistic beliefs nor is there any evidence that they correlate with good clinical performance.

Only those criteria known to predict academic performance, as opposed to clinical performance, in medical school are generally considered in selecting candidates for admission to medical school. In spite of reports by Kelly,[4] Gough, [3, 5] and Korman and his associates[1] criticizing these selection criteria, most medical schools continue to use premedical grades and MCAT performance as the primary selection devices when determining admission to medical school. The logic underlying the use of MCAT scores and premedical grades as primary selection devices is flawed in two ways. The first flaw has already been documented by previously cited literature; that is, MCAT scores do not significantly predict performance on measures of clinical competence collected during medical school. Secondly, measures with which MCAT scores are significantly correlated (i.e., performance on written examinations and academic grades) have not been found to be predictive of subsequent career performance.

This second flaw is substantiated by each of six studies identified by Wingard and Williamson[6] who conducted an extensive review of the literature relating academic performance in medical school with subsequent career performance. Two of these studies[7, 8] used extensive direct observations of physicians' performance measures. Richards and his colleagues[9] used evaluation of letters from hospital officials to rate internship performance. In another study, this group[10] evaluated physician career performance by way of interviews, records, colleagues' opinions, number of journals to which subjects subscribed, and publications. Another group in two studies[11, 12] used board certification and professional appointments or faculty appointments as career performance measures for male graduates. In all these studies little or no correlation was found between grades received in medical school and subsequent career performance.

With the exception of the investigation by Best,[2] studies of the predictive value of the MCAT have used written examinations and supervisors' ratings as criterion measures of clinical performance in medical school. Unfortunately, the subjective nature of supervisors' ratings make them of questionable reliability[13] and supervisors' ratings may largely reflect personality characteristics rather than clinical ability.[1] Thus, supervisors' ratings may not be fair criteria against which to judge the utility of the MCAT for predicting clinical performance. Perhaps MCAT scores can successfully predict other measures of clinical competence, particularly those areas of clinical skill such as interviewing and recognition of psychopathology which require both didactically imparted information as well as more humanistic qualities such as empathy. To date, no comprehensive investigation known to the authors has studied the ability of the MCAT to predict performance of subcomponent skills often subsumed under the rubric of clinical competence. The present study employed five objective measures, in addition to supervisors' ratings, in order to determine the value of MCAT scores for predicting performance on several subcomponent skills of clinical competence in psychiatry.

METHOD

One hundred sixty medical students in their third and fourth years were evaluated using the parameters listed below following a five-and-one-half-week clinical clerkship in psychiatry. Complete data were available for 83 students; therefore, the results are based on a sample of 83.

National Board Examination in Psychiatry, Part II (NBME-II). The NBME-II, though not required, was taken by 83 students at the end of their fourth year.

Written Examination. A multiple choice exam, consisting of 100 items, was administered at the conclusion of the clinical clerkship. Fifty percent of the student's grade was determined by his performance on the test.

Systems Analysis Index for Diagnosis of Basic Psychiatric Syndromes (SAID). SAID, an observational method developed by Miller and Tupin,[14] was used to assess students' ability to identify and grade the severity of pathological behavior. Students viewed a video-taped patient interview and rated 19 symptoms according to severity, using a 7-point scale. Students' scores on the task were determined by comparing their ratings with a criterion rating previously agreed upon by a group of seven "expert" psychiatrists and psychologists (interrater reliability = .93, according to the method of Winer[15]).

Patient Management Problems I and II (PMP I & II). Two PMP's employing a branching erasure format, were used to measure students' diagnostic, therapeutic, and management skills for two common psychiatric disorders, depression (PMP-I) and hyperventilation syndrome (PMP-II). While problems were scored according to the method of McGuire and her associates[16] obtaining efficiency, proficiency and total scores; only the total score was used in the present analyses.

Supervisors' Rating Scale (Super R). Each clinical supervisor rated each of his students on a 9-item Supervisors' Rating Scale using a 9-point scale for each item. Items included time commitment to the learning process, chartwork and patient write-ups, quality of supervision contact, descriptive diagnostic ability, psychodynamic understanding, applied knowledge of psychopharmacology, attitudes toward patients and initiative and thoroughness in patient evaluation. Each supervisor also rated the student's off-service summary, which served as an index of the student's abilities for case formulation and written communication.

Interview Rating (Inter R). Students submitted an audio-taped recording of a patient interview they had conducted. Two psychiatrists residing in another city rated the audio-taped interviews using a 33-item rating form which measured the adequacy of the interview content and interviewer style.

RESULTS

Correlations between MCAT scores and each measure of clinical performance were computed (Table I). Scores on the Total Score and the Verbal and Science sections of the MCAT were significantly correlated with the NBME-II-Psychiatry scores (r = .456, r = .350, r = .357, respectively, P < .001). Similarly, MCAT Quantitative and General Information scores were significantly correlated with NBME-II-Psychiatry (r = .258, r = .339, respectively, P < .01). No other correlations were significant at or below the .01 criterion.

TABLE I

CORRELATIONS OF MCAT SCORES WITH CLINICAL PERFORMANCE MEASURES

(N = 83)

	Verbal	*Gen. Info.*	*Quant.*	*Scien.*	*Total*
NBME-II	.350**	.339*	.258*	.357**	.456**
Exam	.153	.090	.134	.208	.205
SAID	.019	.076	−.087	.005	.007
Inter R	−.109	−.017	.099	−.160	−.039
Super R	.086	.047	.247	.039	.141
PMP I	−.044	.003	.166	.167	.099
PMP II	−.084	−.039	−.022	.013	−.046

* P < .01
** P < .001

COMMENTS

In the present study, MCAT Verbal, General Information, Quantitative, Science and Total scores were significantly predictive of performance on the National Board Exam — Part II in Psychiatry. MCAT performance was not significantly related to performance on various measures of subcomponents of clinical competence including interview ratings, supervisors' ratings, patient management problems, and ratings of observational skills (SAID). The results of the study are consistent with other findings reported in the literature.[1, 2] While MCAT scores appear to be closely related to performance on objective tests of didactic material, their poor prediction of other measures of clinical skills may be more important. The need for scientific ability in the practice of medicine is evident. However, clinical ability appears to be a function of additional attributes, and it is possible that current medical school admission policies are overemphasizing scientific ability to the detriment of clinical competence. In light of recent social developments such as the public's insistence on accountability in educational and health care systems, the increasing numbers of litigations against medical practitioners, and the recent U. S. Supreme Court Bakke decision, medical schools may be obliged to review admission policies with the goal of selecting students with greater diversities of abilities. □

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WARNING: Because of the potential hazard of nephrotoxicity and ototoxicity due to neomycin, care should be exercised when using this product in treating extensive burns, trophic ulceration and other extensive conditions where absorption of neomycin is possible. In burns where more than 20 percent of the body surface is affected, especially if the patient has impaired renal function or is receiving other aminoglycoside antibiotics concurrently, not more than one application a day is recommended.

When using neomycin-containing products to control secondary infection in the chronic dermatoses, it should be borne in mind that the skin is more liable to become sensitized to many substances, including neomycin. The manifestation of sensitization to neomycin is usually a low grade reddening with swelling, dry scaling and itching; it may be manifest simply as failure to heal. During long-term use of neomycin-containing products, periodic examination for such signs is advisable and the patient should be told to discontinue the product if they are observed. These symptoms regress quickly on withdrawing the medication. Neomycin-containing applications should be avoided for that patient thereafter.

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ADVERSE REACTIONS: Neomycin is a not uncommon cutaneous sensitizer. Articles in the current literature indicate an increase in the prevalence of persons allergic to neomycin. Ototoxicity and nephrotoxicity have been reported (see Warning section).

Complete literature available on request from Professional Services Dept. PML.

HEALTH SYSTEMS AGENCIES AND PHYSICIANS' PARTICIPATION*

LYN BEASLEY, M.S.**
J. GAVIN APPLEBY, M.D.***

Physicians can get a better understanding of HSA by considering essentially three areas, the first being an overview of the requirements of Health Systems Agency Board members, generally; secondly, the specific duties of an HSA Board member; and finally, the specific needs of HSAs for physician input and how the special expertise of physicians can be utilized within the HSA structure.

Public Law 93-641 requires that an HSA governing body be representative of the population of the Health Service Area in which it is located. As such, an HSA Board of Directors must be a very heterogeneous group. First, the law requires that a majority of the board be consumers of health care. Among the provider group, the law requires five different types. These types include health care professionals, such as physicians, nurses, other health care professionals that actually provide services to the people. The second type of health care provider required to be represented on an HSA Board are representatives of health care institutions. The third type includes health care insurers. The fourth are representatives of health professions schools, and the fifth type includes allied health professionals.

The consumer portion of the Board of Directors is required to be representative of the area's population in terms of five factors: (1) economic representation; (2) linguistic and racial populations; (3) geographic areas in the Health Service Area; (4) major purchasers of health care and (5) representatives of the social characteristics of the area (male/female, elderly, etc.) The philosophy embodied within this Federal Act is that all views of the community, through its representative Board of Directors on the HSA are needed to plan and develop a total health care system to best meet the needs of the area collectively. This heterogeneous group brings different types of expertise to the HSA Board table for the deliberations of the group. For the most effective HSA activities, the governing body must act as a unit given their different perspectives, abilities, both as community members as well as technical abilities, such as possessed by physicians and other health care providers. This unit or team approach requires that egos and feelings must be put aside if the HSA is to achieve a healthier population and a health care system which will best meet the needs of the people, and I mean true needs of the people, as efficiently as possible. This does not mean that greater efficiency should be accomplished at the cost of reduced quality.

A well-run and effective Health Systems Agency requires a huge amount of time and dedication from all of its Board members. About a year ago in Palmetto-Lowcountry Health Systems Agency, it was estimated that, collectively, Board member volunteers were spending some forty hours per week on the work of the Health Systems Agency. As physicians, active participation requires an understanding of and commitment to these facts, and local medical societies should strive to select from among their membership persons who can commit the time necessary for effective input. Additionally, some bylaws require that a Board member also serve on at least one standing committee of the Board. Most standing committees meet on a monthly basis. Board members who "do their homework" before they get to the meeting are the most effective.

Good planning by the Health Systems Agencies provides for an alternative to more federal control of the health system. Good health planning requires good physician input. There is no such thing as good hip-pocket planning based on pre-conceived ideas and emotions. Good health care planning requires us to put our emotions aside and deliberate over facts to improve local health care problems through local community solutions.

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*** President, Palmetto-Lowcountry Health Systems Agency

What are the functions of the Board of Directors of an HSA? They are responsible for the effective management of the affairs of the agency in such a way as to carry out the missions of the organization in line with their own adopted policies and procedures. The Board of Directors is responsible for the development and adoption of a long range Health Systems Plan and an Annual Implementation Plan. They are responsible for establishment of agency policies. They are required to employ an executive director and charge that person with the responsibility of administering the affairs of the agency. This limits the role of the Board to setting policy as it relates to all other staff of the agency. The Board of Directors is responsible for the authorization of all necessary committees, task forces, the development of special studies, setting agency priorities, etc. It is the responsibility of individual Board members to communicate to the community at large the agency's goals, methods and recommendations. The Board is responsible for the approval of the agency budget. It is required to issue an Annual Report of its activities and accomplishments. It makes recommendations to the Governor for appointments to the Statewide Health Coordinating Council. The committees and Board are *frequently* required to make difficult and quite controversial project review decisions as they relate to Certificate of Need and Federal funding applications. It is also important to note that since the Board of Directors is responsible for Board policy, it is also liable for agency activities. This should not present a problem as long as the Board operates within its own policies and procedures and makes decisions based on informed judgement (based on the facts) and acts in good faith.

How can you, as physicians be most helpful to the HSAs and what are the benefits to medicine from their active participation? There are several areas in which their technical input can further enhance the credibility of the local health planning process. Health planning is the primary focus of the HSA. The plans developed should serve as the policy basis by which all decisions of the agency are made. The plans of all HSAs must get more specific and must address the real world problems and priorities within each area. HSAs use numerous types of committees and task forces for input into the development of the plan. Local medical societies should get on the mailing list to receive draft plans, organize committees within the local medical society to offer positive input into specific areas of the plan. Physicians must *take the time* and *make the effort* to assist the HSAs in making the health plans better. The standards, criteria and policies established in the health plans serve as the basis for project review. Physicians, as well as other health care providers, must make their thoughts and priorities known in the development and setting of these policies. Their technical and objective input should be directed to areas of quality assurance, specified quantities of health service need by the population, particularly as it relates to the national guidelines that were published last year which generated over 55,000 comments to DHEW. A process exists whereby the individual HSAs can adjust the national guidelines to be realistic at the local level because of the uniquenesses and special problems encountered within a Health Service Area. This must be done based on facts and physician input is absolutely necessary.

Physicians must become increasingly aware of the cost impact of their medical decisions as they relate to tests ordered, admissions to inpatient facilities, prescriptions ordered, etc. Physicians need to collectively and critically review the impact of new medical technologies for improved patient care, as well as cost effectiveness. Will the new medical technology replace more traumatic and costly procedures than what are presently being utilized?

It appears that in the very near future, Health Systems Agencies will be reviewing and approving or disapproving grant application requests for federal funding for health manpower education and maybe even in the area of health research. These two areas, more than any other, must have your technical input. We must ask questions such as: Will the program produce health care professionals that are most needed in the community or will it rather cause an excess of existing health manpower? Will the curriculum better prepare the professional to practice good medical care to the patient at the local community level? Who else, better than physicians having been through at least eight years of college education,

(Continued on Editorial Page)

knows of some of the inadequacies of that medical education in preparing the individual to practice medicine? What needs to be beefed up in the curriculum? What needs to be removed from the curriculum? The HSA, as an entity responsible for providing a public forum at the community level, must have physician input in these critical areas.

In summary, HSAs *want* and *need* physicians' technical and objective input. Physicians must take the time and make the effort to help improve HSAs. ☐

ERRATUM

In the June issue, in the article by Dr. George E. Gifford, Jr. on John James Audubon, the painting of the snowy heron in Figure 6 was incorrectly attributed to Lehman, rather than Audubon. Lehman gets credit only for the background.

Editorials

PENICILLIN RESISTANT GONORRHEA

Two patients with gonorrhea which did not respond to penicillin therapy were reported in the August 27, 1976 issue of Morbidity and Mortality Weekly Report (MMWR) from the Center for Disease Control (CDC). The isolates of *Neisseria gonorrhoeae* from these patients were unique because they were the first strains isolated in the United States which produced the enzyme penicillinase (Beta-lactamase). This enzyme provides the organism with the ability to chemically inactivate most penicillins including penicillin G, ampicillin and carbenicillin. Such organisms are referred to as penicillinase producing *Neisseria gonorrhoeae* (PPNG).

When the problem was identified in the United States, the Bureau of Laboratories of the South Carolina Department of Health and Environmental Control (DHEC) screened every isolate of *N. gonorrhoeae* received during the period of September-December 1976 for evidence of penicillinase production. Over 2,400 isolates were tested and none were found to be PPNG.

To assess the prevalence of PPNG in South Carolina, the Bureau of Laboratories is presently examing all positive, post-therapy cultures of *N. gonorrhoeae* using the chromogenic cephalosporin test for penicillinase production. From January 1977 until the present (June 1979), 900 positive post-therapy cultures were examined. None of these organisms were found to be PPNG. During this time (the three year period ending February 1979), the CDC identified 554 isolates of PPNG from patients in 31 of the United States, including Alaska and Hawaii. All index cases were infected in Southeast Asia.

With the advent of PPNG, it is essential that post-therapy cultures are obtained from each patient. Every *N. gonorrhoeae* culture received in this laboratory with the request form (#1325) marked "Test of Cure" is tested for PPNG. We plan to continue this policy of surveillance as long as the threat of penicillin resistant gonorrhea persists.

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SOUTH CAROLINA MEDICAL ASSOCIATION

AUXILIARY

ANDERSON COUNTY AUXILIARY MEETING

The Anderson County Medical Auxiliary has had an exciting and progressive year under the leadership of President Mrs. DeWitt Niles (Jean). At the May meeting, husbands were honored guests at a covered dish supper. Charlestonian, Mrs. Warren Atkins (Skippy) was guest speaker for the evening and presented a program of startling facts on our National Health Insurance Plan. Skippy's talk was entitled "The Potential of the Political Atmosphere and its effect on Medicine." It is our responsibility as citizens to write our Congressman concerning any medical bills currently before the Legislature. Congress currently has before it several Legislative items pertaining to medicine. They are: Health Planning Act, Hospital Cost Containment, The Talmadge Medicare-Medicaid Reform and Reimbursement Act, Long Catastrophic Insurance Act, and National Health Insurance.

Skippy discussed these motions with the Auxiliary. Each member left that evening feeling that we must all become more aware of medicine on a national level and do our part to have some voice in the final outcome. It is up to us to speak out through mail correspondence or local newspapers.

The evening concluded with a covered dish supper and a chance for the Auxiliary members and their spouses to visit with Dr. and Mrs. Atkins.

Anderson County Medical Auxiliary President, Mrs. DeWitt Niles addresses the group at their May supper meeting.

Guest speaker, Mrs. Warren Atkins, speaks on National Health.

President's Pages

PRIORITIES II

In concert with the Council and staff, we expanded our priorities from the original ones discussed on this page last month to include these here. These will guide our activities for the year.

GREATER EFFECTIVENESS WITH REGARD TO PUBLIC RELATIONS:

Improved press and public relations: If the positive position of organized medicine is to be made known to the public, it must be done by organized medicine. Too often negative points are expressed by others without adequate rebuttal. It has been said that things are not necessarily what they are but what they seem to be. Let us, then, express our views and tell our story so that what seems to be is actual.

Our South Carolina Medical Association Committee on Public Relations, under the Chairmanship of Dr. Leon Banov, received approval for the printing and distribution of a brochure telling the SCMA story. The Horry County Medical Society has an on-going public relations campaign which gives visibility to the Society and assistance to the public. Their effort should be studied by other societies.

We have established a list of SCMA spokesmen (geographically located) for referral to the news media when SCMA opinions on health issues are requested.

Continued involvement with governmental agencies and other associations: These agencies have a large impact on physicians' practices, and include DSS, DHEC, the State Board of Medical Examiners and the S. C. Commission on Higher Education. The Council and Executive Committee will serve as a liaison group for these particular bodies. Liaison with professional associations such as the Bar Association, the Hospital Association and the Pharmaceutical Association would more appropriately be assigned to and handled by one of the newly organized Commissions and/or appropriate standing committees.

Investigate private means to fulfill needs in the underserved areas of the state, thus avoiding being preempted by federal programs: At the present time, there are 31 rural and community health centers established in South Carolina. These centers are established by federal funds to meet what is considered to be unmet needs. Five of these centers receive reimbursement for Medicare and Medicaid at an estimated average rate of $25.00 per encounter. This rate is several times greater than the same agencies will reimburse a physician or other health professional for delivering identical care to the same beneficiaries. We find it difficult to encourage physicians to establish private practices in these areas under these conditions. How can we address the issue of maldistribution of physicians?

CONTINUING LEGISLATIVE SURVEILLANCE:

The SCMA will continue to monitor new legislation and amendments to existing legislation and lobby for its position. The "Legislative Update" will be mailed to all South Carolina physicians on a regular basis to keep them informed.

INCREASED MEMBERSHIP IN SOCPAC:

Very few people have independent economic wherewithal to conduct campaigns for election to public office. If we are concerned about legislation, then we must be concerned about who holds office. We must elect qualified people. SOCPAC is a non-partisan group rendering support to the best qualified regardless of party. Your support is urgently needed.

BROADEN SCMA INVOLVEMENT IN CONTINUING MEDICAL EDUCATION:

Council, as designated by the House of Delegates, has recently approved a one-year pilot study of SCMA involvement in CME as a coordinating statewide agency. It is not our intention to be a provider of CME courses, but to coordinate CME efforts, to recommend topics based on physicians' needs, and to be the record-keeping agency of CME credits for the physician.

MANPOWER STUDIES OF PHYSICIANS IN EACH SPECIALTY IN SOUTH CAROLINA:

This project is presently under study by the SCMA Specialty Society Advisory Commission to Council and the S. C. Commission on Higher Education.

Physician supply, based purely on population, does not adequately address the need. We feel that the specialty societies should have input into the determination of that need.

DEVELOPMENT OF A POLICY MANUAL FOR THE SCMA:

We are recommending, after discussion by Council, the development of a policy manual for the SCMA which will include statements of policy established by the SCMA Council and House of Delegates on major issues.

These priorities are established to set our course for this year's activities. Most will be implemented by the officers, councilors and staff, but without full support from the various component societies, the effort needed to succeed will be made much greater.

Let me list a few important items you, as a member, can do — attend your county and district meetings — become involved. Make it your responsibility to enroll at least one new member. Join SOCPAC. Join AMA. Learn something about the people you and your patients are sending to Columbia and Washington to make the rules and regulations you will be expected to live by. You might be surprised to find that these people want to hear your views.

Let's improve our public relations: "Your action speaks so loud, others cannot hear a word you say."

Harrison L. Peeples, M.D.,
President

"Better Health Through Health Education"

1979-1980 SOUTH CAROLINA MEDICAL ASSOCIATION COMMITTEES AND COMMISSIONS

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SYMPOSIUM — PERSPECTIVES IN CARDIOLOGY

BRUCE W. USHER, M.D.*
GERMAN LIZARRALDE, M.D.**

INTRODUCTION

The impetus for this symposium can be traced to 1973-1975 when one (BWU) of us was practicing primary care in a small community in South Carolina. It became obvious during that two year period that one source of frustration to many physicians in private practice in rural South Carolina is the lack of time and resources to keep abreast of current advances in medical care. More importantly was the inability to adequately research the literature in order to place into proper perspective the impact various new advances should have on one's practice. Although we are unable to discuss all of the new entities, concepts, procedures or diagnostic tests pertaining to cardiology, we have made an attempt to select topics which we feel are important to the practicing physician.

The lead article deals with one of the most common but least understood cardiac abnormalities with which the physician is confronted almost daily. Dr. Hendricks and Dr. Usher review the syndrome of mitral valve prolapse and provide current recommendations for management of patients with this syndrome. In the second article, Dr. Larry Kibler summarizes the role as well as limitations of treadmill stress testing and discusses the place of nuclear medicine studies as an adjunct to treadmill stress testing.

As the more recently trained physicians enter private practice in the community hospitals, there will no doubt be an increasing demand for hemodynamic monitoring of the acutely ill patient. Dr. Michael Assey not only provides important tips for those individuals skilled in insertion of Swan-Ganz catheters, but also discusses the rationale behind hemodynamic monitoring.

Dr. Grady Hendrix provides us with an update on advances in long term cardiac pacing and pacemaker follow-up systems. In the last article, Dr. William Barnwell discusses the recently revived and hot topic of formal cardiac rehabilitation programs. In the near future we can expect to see an increase in the number of formal cardiac rehabilitation programs in the larger cities of South Carolina. What benefits can be expected from such programs and the basic mechanics of such a program are well outlined in the article.

It is our hope that this symposium will provide the busy practitioner, especially in primary care, some background and an update of these selected topics. We are grateful to *The Journal* for the opportunity to provide this symposium.

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MITRAL VALVE PROLAPSE: A REVIEW

C. W. HENDRICKS, M.D.*
BRUCE W. USHER, M.D.**

With the advent of echocardiography and appreciation of the clinical significance of nonejection systolic clicks, the era of mitral valve prolapse was ushered in. Currently this syndrome may be the most frequently identified cardiac abnormality in women who present to the primary care physician with symptoms of chest pain and palpitations or a history of abnormal auscultatory findings. The purpose of this paper is to review the salient features of mitral valve prolapse (MVP).

Although the recognition of mid systolic clicks dates back to 1887, a cardiac etiology was not proposed until 1961 by Reid.[1] In 1963 Barlow provided angiographic evidence of late systolic mitral leaflet prolapse.[2] The myocardial phase began in 1969 with Engle's observation of inferior wall contraction abnormalities seen on left ventricular cineangiography.[3] The current information explosion related to mitral prolapse can be traced to the advent of echocardiography.

PATHOPHYSIOLOGY

The origin of the click in MVP is dysfunction of one or more of the components of the mitral valve apparatus (valve leaflets, chordae tendiniae, papillary muscles and valve annulus). The click is associated with billowing of the mitral leaflet into the left atrium or tensing of chordae during systole. The murmur of MVP represents mitral insufficiency secondary to coaptation abnormalities of the mitral leaflets. The common pathologic denominator is myxomatous proliferation of the leaflets and chordae.

ETIOLOGY

In the majority of patients with MVP, the etiology is unknown. Speculation has arisen as to whether this syndrome represents a variant of rheumatic fever, a "forme fruste" of Marfan's Syndrome, or represents a form of congenital heart disease. Systolic clicks-murmurs have been documented in acute rheumatic fever, chronic rheumatic heart desease and following mitral commissurotomy.[4] MVP is found in approximately 20% of patients with a secundum atrial septal defect.[5]

CLINICAL HISTORY

The clinical manifestations of MVP are protean. Although many patients with MVP (defined as the characteristic click and/or murmur detected on routine physical exam) are asymptomatic, the majority of patients present with chest pain, palpitations, dyspnea and fatigue.[6] These symptoms are frequently interpreted as "neurotic complaints." Occasionally, syncope and neurologic events including transient ischemic attacks[7] and migraine[8] can be elicited.

The chest pain is usually atypical for angina. A pressure quality may be ascribed to the discomfort, but an exertional association is unusual. The discomfort may be sharp and stabbing, lasting from minutes to hours. The pain can occur anywhere in the precordium, and the discomfort can occur in different locations at various times in the same patient. The intensity can vary from trivial to incapacitation.

Palpitations occur commonly with MVP. This symptom can be prominent even during normal sinus rhythm. Supraventricular as well as ventricular tachyarrhythmias occur with MVP, hence, palpitations may the be harbinger of a malignant rhythm disturbance.

Dyspnea and fatigue are problems of a nonspecific nature which may by the sole complaint.

PHYSICAL EXAM

In addition to the typical auscultatory features, other physical findings should be looked for when considering MVP. Many subtle findings can de discovered: musculoskeletal abnormalities including straight back, pectus deformities, scoliosis, asymmetry of the chest wall, and Marfanoid habitus. This association is explained by the fact that the primordia of the mitral valve undergoes differentiation between the 35th and 42nd days of fetal life, the same time that the vertebrae and thoracic cage are beginning chondrofication and ossification.

---

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Palpation of the precordium may reveal a bifid impulse which corresponds with mid-systolic retraction of the ventricle coinciding with the peak of the mitral valve prolapse.

The hallmark of MVP remains cardiac auscultation. Clicks alone may be heard in 27%, typical murmur alone in 26%, and click with murmur in 22%.[7] The absence of auscultatory findings does not rule out MVP. The click is usually a mid to late systolic sound. The murmur may be holo, mid or late systolic, is usually crescendo, and it may have a "honking" or "Whooping" characteristic. When both click and murmur can be heard, the latter usually begins with or occurs shortly after the click.

Bedside maneuvers can be used to alter hemodynamics and therefore the timing in systole of the click and murmur. A decrease in the left ventricular volume (sitting, standing, Valsalva, amyl nitrite inhalation), allows more redundancy of the valve leaflets and chordae resulting in an earlier click and murmur. An increase in left ventricular volume (supine position, squatting, Inderal, and pressor agents) minimizes the chordal and leaflet redundancy and the timing of the murmur and click will be later in systole. These simple maneuvers may unmask an otherwise "silent" MVP. Many clicks and murmurs are very soft, and careful auscultation should be performed in multiple positions when MVP is suspected.

X-RAY

The most common roentgenographic abnormalities are the associated skeletal abnormalities. The cardiac silhouette is generally normal.

ECG

Electrocardiographic abnormalities are common. Routine ECG's, exercise stress testing, and ambulatory Holter Monitoring are all useful in evaluating these patients. Routine electrocardiograms may reveal prolonged QT intervals, uniform or multiform PVCs, bigeminy and occasionally ventricular tachycardia. First degree AV block has been reported in about 3% of patients.[4] Ten-fifteen percent of patients referred for refractory ventricular tachycardia are found to have MVP as the sole etiology. Atrial premature beats, junctional premature contractions, sinus tachycardia, paroxysmal atrial tachycardia, atrial fibrillation and atrial flutter are not uncommon. ST and T wave changes may occur with T wave inversions in the inferior leads being most frequent. Prominent Q waves may be seen in the inferior leads and when such findings occur in young females, MVP should be suspected.

Exercise testing is useful in evaluating the arrhythmias found in MVP. Baseline ectopic ventricular activity usually abates as exercise progresses; however, in the immediate post-exercise period malignant arrhythmias may occur.

The most sensitive monitoring modality is the 24 hour continuous ambulatory recording. Both atrial and venticular arrhythmias can be recorded and correlated with a patient diary. This can be useful in patients who have frequent dizziness and palpitations. Repeat stress testing or Holter Monitoring is useful in determining therapeutic benefit of antiarrhythmic therapy.

ECHOCARDIOGRAPHY

Much of the literature on MVP stems from the advent of echocardiography. This noninvasive procedure will be diagnostic or suggestive of MVP in 70-90%.

Echocardiography is usually the means by which the diagnosis is made in patients who have no clicks or murmurs and it is usually confirmatory when clicks or murmurs are heard. The echocardiogram should not be considered an absolute essential when the classical click and/or murmur are present. If one is not certain of the auscultatory findings, an echocardiogram should be obtained since ejection clicks (pulmonary and aortic in origin) and even opening snaps (mitral-stenosis) can be confused with the click of MVP. An echocardiogram may be of help in evaluating the patient with arrhythmias and/or atypical chest pain and normal auscultatory examination.

CARDIAC CATHETERIZATION

Cardiac catheterization is reserved for those patients with clinical features of significant mitral regurgitation and those patients with MVP whose pain is typical of angina pectoris.

COMPLICATIONS

There are four well known complications associated with MVP: sudden death, progressive

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mitral regurgitation, bacterial endocarditis and ruptured chordae tendiniae. A fifth possible complication is transient ischemic attacks and cerebrovascular accidents. The most dreaded sequelae of MVP is sudden death, however the accumulated experience with this problem would indicate an incidence of slightly less than 1.5%.[9] The risk of this complication would appear to be greatest in young to middle age females with syncope who have a positive family history of sudden death.

Endocarditis is a significant problem in MVP syndrome. In one series of 62 patients followed over 9-22 years, five developed endocarditis.[10] Therefore, endocarditis prophylaxis is indicated in patients with MVP.

When a patient with MVP suddenly decompensates, a ruptured chordae should be considered. The murmur usually becomes considerably louder and holosystolic. Surgical intervention may be necessary when a ruptured chordae occurs.

Progressive mitral regurgitation is one of the more serious complications also occasionally necessitating mitral valve replacement.

Transient or fixed neurologic deficits have been reported in patients with MVP.[11] The etiology of these neurologic complaints remains unclear although arrhythmias and myxomatous or platelet emboli must be considered.

NATURAL HISTORY AND PROGNOSIS:

Good natural history studies are lacking because of the necessity for long term follow-up and the relatively short period during which this entity has been recognized.

In the young to middle-age group, females predominate. However, the sex distribution is equal at both extremes of life. In general, the prognosis appears excellent. The major complications are rare and if the patient is properly reassured, normal life-style and activities can be maintained.

THERAPY

We currently recommend endocarditis prophylaxis for all our patients with MVP. In individuals with chest pain and palpitations, Inderal is the drug of choice with many patients responding to as little as 10 mgms 2-3 times per day. When supraventricular arrhythmias are found, Digoxin may be added. Quinidine and Dilantin may be used with more complex ventricular arrhythmias that are unresponsive to Inderal. Rare patients with refractory chest discomfort and sustained refractory ventricular tachycardia have been subjected to mitral valve replacement or other operative procedures with variable results. Mitral valve replacement may be required for the patient with moderate to severe mitral regurgitation. The value of physician understanding and reassurance of the patient cannot be over emphasized in order to avoid cardiac neurosis.

SUMMARY

Mitral valve prolapse is a common cardiac disorder affecting all age groups but especially young females. The diagnosis can be made on clinical grounds and confirmed by noninvasive diagnostic techniques. For the majority of patients, therapy is usually simple. Physician recognition and understanding of the MVP syndrome is essential to preclude patients from being labeled as "psychoneurotic" because of atypical chest pain and palpitations. Awareness of the potential complications is essential. ☐

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EXERCISE STRESS TESTING — PAST, PRESENT, AND FUTURE

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The empirical discovery that the electrocardiogram may be altered during coronary ischemia dates to 1918.[1] The value of stress testing for evaluation of coronary artery disease was appreciated in the 1930's following the description of ST and T wave changes during exercise. The Master's Step Test of the 1940's and 1950's gradually began to be replaced by the motorized treadmill, as the more precise level of exercise with determination of aerobic fitness was popularized by Bruce.[2] Normograms have been developed that enable one to determine how well a patient performs for a specified age group, thereby objectively providing an assessment of the degree of functional impairment.[3]

CURRENT CLINCAL USEFULNESS OF EXERCISE TESTING

The most frequent indications for stress testing are listed in Table I.

Studies using the treadmill stress test to evaluate patients with valvular heart disease are limited in numbers. Although a general reduction in indices of left ventricular function have been correlated with a reduction in oxygen comsumption with upright exercise, this kind of exercise has proven somewhat unreliable in predicting the hemodynamic severity of stenotic and regugitant valve lesions in patients subsequently undergoing cardiac catheterization. Further study will be required to determine what exercise parameter, if any, can be used to predict the severity and progression of valvular disease noninvasively.[4]

Currently, the most prevalent use of the treadmill stress test is for evaluation of patients with coronary artery disease. A number of EKG changes have been described in patients with ischemic disease undergoing exercise, but most authorities have accepted 0.1 MV (1 mm) ST wave depression of horizontal or downsloping type for 0.08 seconds as the diagnostic level with the greatest sensitivity without loss of specificity. Recent literature correlating the exercise stress test with coronary angiography in patients having symptoms of angina pectoris as well as asymptomatic subjects is presented in tables II and III. The sensitivity (percentage of patients with disease correctly identified) and specificity (percentage of normal subjects with a negative test) are similar in groups of symptomatic and asymptomatic patients.[11] However, the predictive accuracy of the test, i.e. the likelihood that a positive test is a true positive and therefore indicative of disease, is of more value to the clinician. From tables II and III it can be seen that the predictive accuracy of a positive test is greater (>

TABLE I

INDICATIONS FOR EXERCISE STRESS TESTING

1. Confirmatory evidence for suspected coronary disease and aid in differential diagnosis of chest pain.
2. Prediction of risk of death and coronary events
3. Determination of aerobic capacity
4. Demonstration and determination of behavior of arrhythmias under stress
5. Evaluation of therapy
6. As aid in developing an exercise prescription for the cardiac patient

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TABLE II

Study	*No. Patients*	*Sensitivity*	*Specificity*	*Predictive Accuracy*
Kelemen[5]	74	54%	96%	96%
Goldschlager[6] . . .	330	64%	93%	95%
McNeer[7]	1222	54%	92%	90%

Results of exercise stress test in three recent studies in symptomatic patients undergoing coronary angiography.

TABLE III

Study	*No. Patients*	*CAD*	*Normal*	*Predictive Accuracy*
Froelicher[8]	111	34	60	31%
Borer[9]	30	11	10	37%
Erickssen[10]	75	48	27	64%

Results of coronary angiography in three recent studies of asymptomatic patients with abnormal exercise tests. CAD = Coronary Artery Disease (greater than 50% narrowing of at least one coronary artery).

90%) in the group of symptomatic versus asymptomatic patients (≤ 64%). These predictive accuracy differences can be related to differences in disease prevalence. A positive exercise test in the asymptomatic patients will have a high probability of being a false positive (up to 69%). Conversely, a negative test in the symptomatic patient will have a high probability of being a false negative (43% in McNeer's study[7]), and does not exclude coronary disease.

Despite inherent limitations of the exercise test in the diagnosis of coronary disease, recent studies have shown that much prognostic information can be obtained from the test. In one of the first follow-up studies, Robb and Marks, using the Master step test, found that the death rate over a 10 year period in patients with an ischemic ST response was four times higher than for those with a negative test.[12] More recently, the prognostic value of the treadmill exercise test in symptomatic patients has been confirmed by Ellestad[13] and McNeer.[7] In Ellestad's series, an overall incidence of new coronary events (angina, death, or myocardial infarction) of 9.5% per year in those with significant ST depression compared to 1.7% per year in those with a negative test, increased to 15% per year if a positive test occurred within 3 minutes of exercise compared to 4% per year if the test became positive at more than 7 minutes. In McNeer's series, which had angiographic correlation, exercise into Stage IV or greater was associated with a 48 month survival of 93% and only a 9% incidence of 3 vessel disease, whereas a positive test in Stage I or II dropped the 48 month survival rate to 63% and predicted an incidence of three vessel disease greater than 50% and an incidence of left main disease greater than 24%. The depth of ST depression has been shown to have less prognostic value and a low specificity for critical coronary lesions.[13, 14]

Data from the Seattle Heart Wach[15] indicate that the three variables of cardiomegaly, exercise duration less than 3 minutes, and failure to increase systolic blood pressure above 130 mmHg can identify patients with high sudden cardiac death mortality. In the absence of these three variables the annual rate of sudden cardiac death was 12/1000 men at risk and rose to 638/1000 men at risk in the presence of all three. The most powerful predictor was the maximal systolic blood pressure.

In summary, the exercise stress test may prove to be of value in the selection of patients for invasive testing. It is likely that further definition of exercise variables will aid this selection in the future.

DETECTION OF MYOCARDIAL ISCHEMIA BY THALLIUM 201 SCINTIGRAPHY

In an attempt to improve the diagnostic accuracy of the exercise test in coronary disease, attention has been directed toward various radiopharmaceuticals which are distributed to the myocardium after intravenous injection in proportion to regained blood flow. Thallium 201 has proven to be the best agent for clinical use.[2, 3] The technique most often used to detect myocardial ischemia requires intravenous injection of the agent at the onset of angina or major fatigue while exercising on the treadmill. Exercise is continued to another minute. Immediately after exercise, multiple views of the myocardium are obtained with the scintillation camera. If defects are noted, repeat imaging can be done at rest 4 hours later. Defects on exercise disappearing at rest are consistent with myocardial ischemia. A recent multicenter study[16] using this technique to evaluate patients with suspected coronary disease undergoing coronary angiography demonstrated a sensitivity of 81% and specificity of 86%. The Thallium exercise test seems to be more sensitive than the electrocardiographic stress test, however, a combination of scintigraphy and stress electrocardiography may prove more sensitive than either technique alone.

The Thallium test will likely prove of great value in patients in whom the stress electrocardiogram cannot be reliably interpreted. Such patients are those with left bundle branch block, left ventricular hypertrophy, resting ST and T wave changes, electrolyte imbalance, Wolf-Parkinson-White Syndrome, or those taking digitalis. Also, coronary lesions of questionable significance on angiography can be evaluated for hemodynamic obstruction. One study indicates that in the asymptomatic patient with a positive stress electrocardiogram, a negative scintigram may provide sufficient evidence against coronary disease to avoid cardiac catheterization.[17] It would appear that the stress scintigram will prove to greatly enhance the clinical value of stress testing in the future.

COMPUTER ANALYSIS OF THE EXERCISE ELECTROCARDIOGRAM

Significant difficulties in interpretation of the stress electrocardiogram with large inter and intraobserver variation has been demonstrated. Computer assisted interpretation has been proposed as a way of obviating this error.[18] Computer programs can easily accommodate complex sets of criteria and multiple measurements which may enhance the sensitivity of the stress test. The use of these techniques remains largely experimental, but future work in this field will likely be rewarding. ☐

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THE USE OF SWAN-GANZ PULMONARY ARTERY CATHETERS IN COMMUNITY HOSPITALS

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In an effort to provide a more rational and systematic approach to the diagnosis and treatment of acutely ill patients, physicians in regional medical centers are increasingly relying on invasive bedside hemodynamic monitoring techniques. Knowledge of the systemic arterial, pulmonary arterial, and pulmonary capillary wedge pressures, allows one not only to make more precise diagnoses, but also to confidently make patient management decisions in circumstances where inappropriate or delayed therapy could contribute to the patient's demise. This advantage need not, nor should not, be available only to physicians in large centers, since often the very patient who would maximally benefit from this approach is too ill to be safely transferred.

SWAN-GANZ CATHETER

Bedside catheterization of the right heart became feasible when Swan and Ganz developed the balloon-tipped, flow-directed, pulmonary artery catheter.[1] This catheter can be introduced through a peripheral vein and advanced to a position in the pulmonary arterial tree where the recorded pressure reflects the filling pressure of the left ventricle, one of the major determinants of left ventricular stroke volume. It is no longer necessary to follow potentially misleading right heart pressures (e.g., CVP).[2] The rationale that allows a wedged catheter to measure left heart pressures is that, at the end of diastole, the left ventricle, left atrium and pulmonary capillary bed are functionally a common chamber and therefore, with certain important exceptions, left ventricular end-diastolic pressure = wedge mean pressure = pulmonary artery end-diastolic pressure.

The catheter itself (fig. 1) is 110 cm long, and made of polyvinyl chloride. There are several different types of Swan-Ganz catheters. The standard catheter has two lumens, one which allows pressure determination at its tip through a fluid filled system, and the other through which air is injected to inflate a balloon which encompasses the tip and allows flow-directed movement during insertion. The triple lumen catheter has the same basic structure, but in addition there is another opening thirty centimeters proximal to the tip which usually lies in the right atrium when the catheter tip is in the pulmonary artery. This allows simultaneous measurements of the right atrial and pulmonary arterial or pulmonary capillary wedge pressure. Another type is the triple lumen catheter with a thermistor that allows measurement of blood flow through the heart (cardiac output) by the thermodilution technique using a commercially available computer at the bedside.

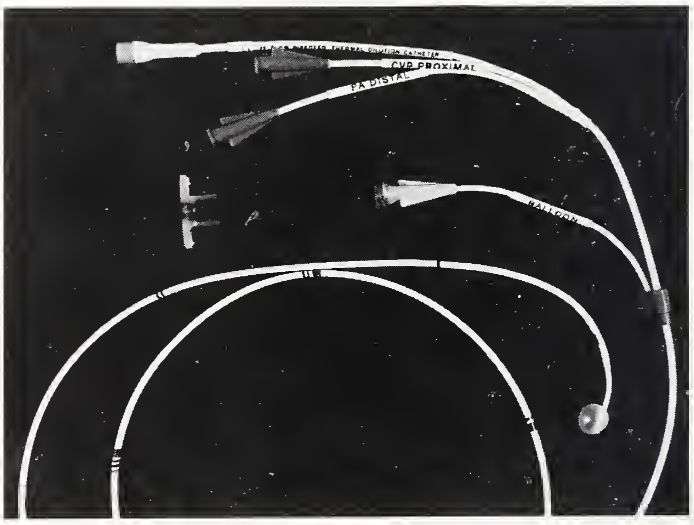

FIGURE 1. Example of Swan-Ganz catheter with thermistor for performing thermodilution cardiac outputs.

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In order to prevent clotting of the catheter and maintain a closed system, a continuous infusion of heparin in 5% dextrose (2000 u/1000 cc) is used. The balloon is inflated with room air (0.8-1.5 cc) unless there is an abnormal access to the left heart (e.g. ASD) in which case more soluble CO_2 should be used to decrease the risk of air embolism should rupture of the balloon occur.

Possible sites of catheter insertion include the antecubital veins, subclavian veins, or internal jugular veins. It is usually more difficult to pass the catheter through the femoral vein without fluoroscopic guidance. A cutdown or percutaneous method can be used for inserting the catheter. Any vein that is large enough is useable, but one should avoid the plump superficial veins of the lateral aspect of the antecubital fossa which drain into the cephalic vein and enter the axillary vein at an acute angle. Flushing of the catheter and checking the balloon for air leakage should precede insertion. Proper catheter position is monitored during insertion by observing pressure wave forms displayed on an oscilloscope through a pressure transducer. When the tip of the catheter reaches the thorax, undulations in the wave form corresponding to respirations are noted. Having the patient cough at this time will result in a marked increase in pressure and assures an intrathoracic position. The balloon should be inflated at this time to minimize arrhythmias during passage through the cardiac chambers, and aid in proper flow direction. During passage through the right ventricle, PVC's are frequently noted. Some physicians routinely administer a 50 mg bolus of i.v. lidocaine just before entry into the right ventricle, but we feel that this is unnecessary as long as the ECG is closely monitored and the catheter immediately withdrawn at the onset of any sustained ectopy. Recognition that one cardiac chamber has been passed through and another entered is marked by a change in contour of the pressure wave (fig. 2), and it is essential that those involved in the placement and/or care of these catheters be able to recognize these various wave forms. As a general rule, if the catheter is advanced within the heart more than 15 cm without a change in pressure wave form, it is likely tha the catheter is coiled in the right atrium or right ventricle, in which case it should de withdrawn to the entry point of that chamber and advanced again. Once a pulmonary capillary wave pressure curve is obtained, the balloon is deflated and a pulmonary arterial wave form should appear. The best evidence of proper catheter position is the ability to easily wedge the catheter with approximately 1 cc of air, and on deflation, a pulmonary arterial pressure wave reappears.

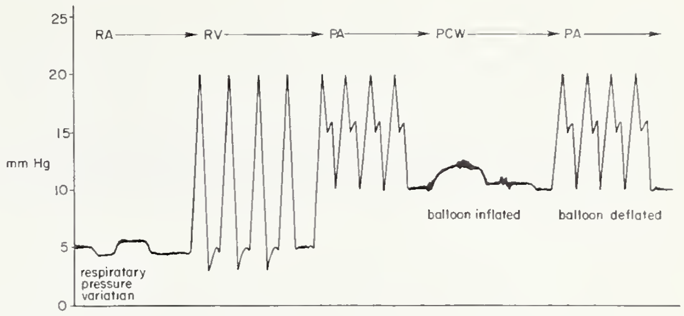

FIGURE 2. Characteristic pressure wave forms as the catheter is advanced from right atrium to the pulmonary artery. RA = Right Atrium, RV = Right Ventricle, PA = Pulmonary Artery, PCW = Pulmonary Capillary wedge.

It is imperative that care be taken to position the transducer at the level of the heart, zero it properly against atmospheric pressure, calibrate it against a known pressure (mercury manometer), and clear the line of any bubbles which are a common source of dampened pressure waves and inaccurate readings.

CLINICAL INDICATIONS

Table I lists circumstances in which the Swan-Ganz catheter has proven to be helpful in patient management and diagnosis.

Table II includes some of the more typical hemodynamic profiles observed, and serve as examples of how the Swan-Ganz catheter aids in patient management.

Case A was a 54 year old man transferred to Medical University of South Carolina twenty-four hours following admission to his local hospital with an acute anterior myocardial infarction. His blood pressure had dropped to 80/60, and his urine output had been only 100 cc during the subsequent 12 hours. His physical examination revealed cool and clammy skin. The lung fields revealed bibasilar rales. Cardiogenic shock was diagnosed, and he was started on dopamine infusion prior to transfer. At the Medical Univeristy Hospital, a Swan-Ganz catheter was inserted and pressures listed in Table II were noted. There was evidence of mild pulmonary hypertension consistent with COPD, but the pulmonary capillary wedge pressure (reflecting the LV filling pressure) was low, indicating that the cause of the patient's hypotension and hypoperfusion was volume depletion rather than left ventricular failure. Following infusion of a liter of normal saline, his pulmonary capillary wedge pressure increased to 12 mm Hg, his blood pressure increased to 116/80 and his urine output increased to 40 cc/hour.

TABLE I

SITUATIONS IN WHICH THE SWAN-GANZ CATHETER MAY BE HELPFUL

1. When there is a need to know the fluid status (left ventricular filling pressure)
    a. acute myocardial infarction with hemodynamic instability.
    b. acute, and sometimes chronic, renal failure.
    c. third space fluid loss.
    d. septic shock.
    e. acute volume depletion, particularly in the patient with underlying cardiac disease.
    f. adult respiratory distress syndrome.
    g. diffuse pulmonary disease.
2. When there is a need to obtain diagnostic information.
    a. new cardiac (holosystolic) murmur (acute mitral regurgitation vs ventricular septal defect).
    b. obtaining mixed venous pO_2 in a patient with "Combined systems" disease (e.g. anemia + low cardiac output, COPD).
    c. right ventricular myocarcial infarction.
3. When intravenous vasodilator therapy is required.
    a. acute, severe mitral or aortic insufficiency, pending surgery.
    b. hypertensive crisis (particularly in patients with coronary disease).
    c. spontaneous idiopathic lactic acidosis, treated with sodium nitro-prusside.
4. Initiation of oral vasodilator therapy as treatment for congestive heart failure in a poorly compensated patient.

TABLE II

Case	*Systemic BP*	*RA*	*RV*	*PA*	*PCW*
	(120/80)*	(2-8)	(12-30/0-8)	(15-30/6-12)	(6-12)
A....	80/60	6	28/6	28/18	4
A+ ..	116/78	10	40/10	40/28	12
B....	80/50	16	38/16	38/16	16
C ...	100/85	6	30/6	30/22	22

* parentheses include normal values in mmHg.
+ following therapy

This case illustrates a key value of hemodynamic monitoring. The physical examination, because of coincidental lung disease, was misleading and therapy that resulted was detrimental (i.e., the sinus tachycardia induced by vasopressor infusion in a volume depleted patient increases mycocardial oxygen demand). After volume expansion the pressure revealed pulmonary hemodynamic profile was recorded (B, Table II). The "equilibration of diastolic pressures" resulted in this case from pericardial tamponade, capillary wedge pressure because of intrinsic pulmonary vascular disease, and one would follow the wedge pressure, not the pulmonary artery diastolic pressure, as a guide to fluid expansion.

Case B, was a 26 year old woman with chronic renal failure who was hospitalized with increasing shortness of breath, peripheral edema and distended neck veins. The admitting diagnosis was "volume overload and biventricular failure." Because of progressive hypotension, a Swan-Ganz catheter was inserted, and a characteristic hemodynamic profile was recorded (B, Table III). The "equilibration of diastolic pressures" resulted in this case from pericardial tamponade, a situation in which the intrapericardial pressure becomes the determinant of intracardiac diastolic pressures. Pericardiocentesis resulted in immediate hemodynamic and symptomatic improvement, and the patient was managed with more frequent hemodialysis.

Case C was a 55 year old man who was stable five days after a myocardial infarction. While on the ward, he developed acute dyspnea and orthopnea, and physical examination revealed a new harsh holosystolic murmur at the lower left sternal border. A triple lumen Swan-Ganz catheter was inserted, and in addition to the pressures recorded (consistent with acute left ventricular heart failure), blood samples were drawn from the right atrium and pulmonary artery. The oxygen saturation in the right atrium was 62%, but in the pulmonary artery was 84% which helped identify the cause of the new murmur as a ruptured interventricular septum with resulting left to right intracardiac shunt.

The use of Swan-Ganz catheters is not without risk and include local and systemic infection, transient arrhythmias, pulmonary thromboemboli and infarction, mostly as isolated case reports. Still, the benefit to risk ratio in a properly selected population is extremely favorable, and physicians in community hospitals should avail themselves of hemodynamic monitoring to maximize patient care. ☐

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ADVANCES IN LONG TERM CARDIAC PACING AND PACEMAKER FOLLOW-UP

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The advent and development of long term cardiac pacing has been one of the major technical advancements in managing cardiovascular diseases. Individuals with conduction disease only are restored to a normal life expectancy with implantable pacemakers.[1] Stokes-Adams attacks, the most common indication for pacemaker implantation, are totally eliminated with effective long term pacing.[2] A variety of arrhythmic disorders as well as cardiac decompensation can be more effectively managed with the cardiac rhythm under the control of a pacemaker.

There have been four major breakthroughs since pacing was observed to be possible. These are:

1. The development of an implantable pacing device.
2. The transvenous approach to pacing.
3. Introduction of demand pacemakers.
4. The development of the long term non-nuclear, non-rechargeable power source.

The following will be an elaboration on recent advances and technological changes as well as emerging concepts in long term pacing.

POWER SOURCES

The Lithium-Iodide power source has rapidly become the preferred mode in virtually all pacemakers. Longevity at the present time is predicted to be 6-12 years. The Mercury-Zinc power source, the backbone of permanent pacing until Lithium-Iodide became available, should not be used for anyone except patients with a life expectancy of only 2-4 years. There is a two cell Lithium-Iodide pulse generator available at a comparable cost to the Mercury-Zinc systems which is replacing it even in that group of patients. Nuclear source (Plutonium and Prometheium) have virtually disappeared from the scene due to the advent of Lithium-Iodide power sources. When one considers that the life expectancy of males after pacemaker implantation is 10.6 years and females 13.4 years, it appears that most patients will have none or one pulse generator replacement with the presently available Lithium-Iodide Pacemakers.[3] The immense degree of governmental regulation also prevented development and use of the nuclear power source. The Nickel-Cadmium rechargeable power source failed to gain patient acceptance due to the weekly recharging schedule necessary.

PROGRAMMABLE PACEMAKERS

The ability to non-invasively change an implanted pulse generator's output parameters will be a major advancement and is on the threshold of widespread use. Experience was gained in this area invasively 10 years ago but it was discontinued due to problems associated with the invasive technique. Presently available are two models in which output rate can be changed non-invasively; one has rates of 60, 65, 70, 80, 90, and 100 per minute and the other 50, 60, 65, 70, 100, and 120 per minute. Most patients get along well with a rate of approximately 70 per minute but some advantages of being able to vary the rate are:

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1. The control of the pain of ischemic heart disease may be much better at a rate of 50 than 70, particularly in patients receiving propranolol therapy.
2. Some patients suffer hemodynamic embarassment when paced and pacing may be less necessary if it occurs at a rate of 50 or 60 rather than 70. This may be quite feasible in patients with sinus node disease.
3. Patients may become ill from other sources and require an increased rate to cope with fever, anemia, etc.

These pacemakers are also programmable to vary output at levels of 4.5 volts, 3.0 volts, 2.0 volts, and 1.15 volts. Assuming an impedance of 500 OHMS the milliamps would be twice these figures. The output can be reduced to the lowest level needed for pacing after implantation and when the maximum resistance has been reached, thereby prolonging the life of the pulse generator as opposed to the fixed output of 4.5 volts of the non-programmable pacemakers.

Programmable pacemakers appear to be technologically sound and cost only slightly more than the non-programmable types and no doubt will eventually be used routinely for all implants. Work is going forth on developing pulse generators in which the pulse duration, sensitivity and refractory periods can also be programmed non-invasively.

LEAD SYSTEMS

1. Transvenous: the transvenous route of implantation remains the dominant one with approximately 80% performed by this route. Pacing analyzers are available which determine the threshold of stimulation, amplitude of the R wave, and signal frequency of the endocardial electrogram and this should always be performed to determine if satisfactory pacing and sensing can be derived at the endocardial site. The preferred cutdown site remains the cephalic but many physicians now prefer to place the lead into the subclavian vein percutaneously via an introducer. An introducer cannot be used for the endocardial leads which have anchors or tentacles. A number of electrodes are now available which grasp the right ventricular endocardium to prevent dislodgement. The "Tyne" tip has four silicone rubber tips which anchor it in place and seems to be safe and effective. Other types available have nylon wires, metal wires, a "screw-in" endocardial electrode, and a balloon tip. My feeling is that if the standard and "Tyne" tip fail to hold position, an epicardial lead should then be performed.
2. Epicardial: The evaluation of lead implantation has gone through myocardial via thoracotomy to transvenous with a minimum risk with a subcostal epicardial implantation as an alternative. It does appear that implantation of the sutureless electrode on to the right ventricular epicardium is not as desirable because the signal for the demand mode of the pacemaker arises from the left ventricle and may be inadequate to trigger a right ventricular placed pulse generator.[4] The electrodes can be implanted onto the posterior left ventricular epicardium via a left subcostal incision.

PACEMAKER FOLLOW-UP

The two major modes of pacemaker follow-up are to see the patient at frequent intervals or to see the patient at infrequent intervals combined with transtelephonic transmission of the ECG. It is recommended that the system be monitored at weekly intervals for four weeks after implantation of a total system. This is not necessary when pulse generator replacements only have been carried out as any failure during the first month is usually a lead problem.

The subsequent follow-up for Mercury-Zinc pulse generators is as follows:[5]

2 - 18 months	Every 2 months
19 - 24 months	Every 1 month
24 - 30 months	Every 2 weeks
Over 30 months	Weekly

The subsequent follow-up schedule for Lithium-Iodide pulse generators remains unclear as such information is not yet available. One present manufacturer's recommendation is monthly which is our schedule.[6]

The transtelephonic transmission system enables patients to have a transmitter at home which they can use to call a central location where an ECG strip can be recorded and analyzed. It appears safe for the patient to induce fixed rate pacing with an external magnet for a telephonic pacemaker check. This transmitter can be purchased with the pacemaker when hospitalized for implantation.

We use the above described schedules for telephonic transmission. The patient is seen in our pacemaker follow-up clinic or by his primary physician, internists, cardiologists, or cardiac surgeon one month after implantation and then yearly unless dictated more frequently by non-pacemaker problems. A simple device is available for measuring pulse width when the patient is seen. This adds some information on pacemaker longevity but not enough at the moment to be universally useful and dependence on rate changes appears to be adequate follow-up.

ATRIAL PACEMAKERS

Most patients with SA node problems have been treated with ventricular pacing due to the unreliability of atrial pacing and sensing and the necessity for a major thoracotomy implantation. Pacemakers are now available which satisfactorily sense the atrial electrogram, pace satisfactorily, and can be implanted transvenously into the atrial appendage. A His Bundle study should be performed on all patients before implantation of an atrial pacemaker as any covert AV conduction problems would preclude an atrial pacemaker.

ATRIAL TRIGGERED VENTRICULAR PACEMAKERS

A transvenous model of this type of pacemaker is now available but is technically difficult and it appears that a major thoracotomy is the best route for its implantation. It remains a good pacemaker choice for active young individuals.

AV SEQUENTIAL PACING

The desirability of maintaining synchronous atrial and ventricular contraction is important in some individuals, particularly those in heart failure. This pacemaker, which paces the atrium followed by a normal PR interval and then paces the ventricle, is available for implantation by the transvenous or epicardial route. The technical considerations of transvenous implantation remain major.

UNDER DEVELOPMENT

1. The detection of and termination of supraventricular re-entrant arrhythmias by a train of stimuli.
2. The detection of and termination of ventricular tachycardia or fibrillation with an implanted defibrillator.
3. A built in device in the pacemaker which could detect malfunction and signal the patient.

SUMMARY

The present status of implantable pacemakers for long term pacing and modes of pacemaker follow-up has been reviewed. The new generation of pulse generators with the Lithium-Iodide power source appears to be a major advance over Mercury-Zinc power sources and have virtually supplanted them as well as aborted the advent of Plutonium power pulse generators. Eighty percent of implants are now performed by the transvenous approach and the emerging generation of transvenous leads which anchor to the right ventricular apex no doubt will continue to make this route the preferred one. Transtelephonic pacemaker monitoring from the patient's home to a central facility has developed to the point of reliable ECG strips being readily available for interpretation by a physician. ☐

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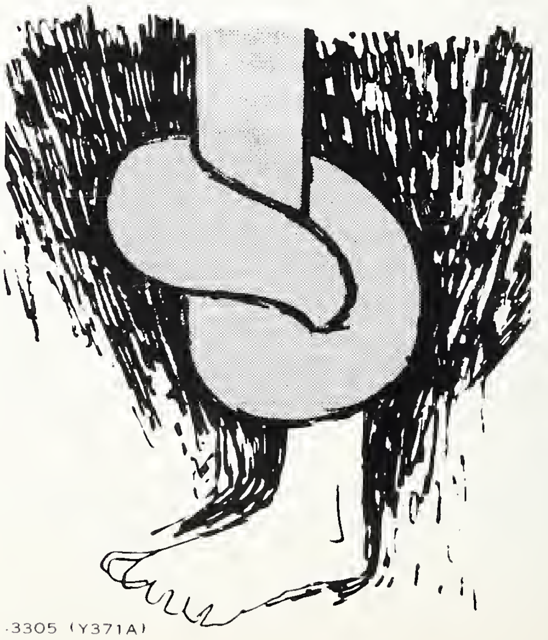

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CARDIAC REHABILITATION

WILLIAM H. BARNWELL, M.D.*

CARDIAC REHABILITATION

Cardiac rehabilitation has become a common term only within the last two decades but dates back about twice that span. An early step was the development of Work Evaluation Units, the first by Leonard Goldwater, M.D., in 1945. Major conferences on this subject have been held in Wisconsin, 1959, in Myrtle Beach, S. C., 1969, and the first international conference in Hamburg, Germany, 1977, and their proceedings published. The American Heart Association Rehabilitation Subcommittee issued a Cardiac Rehabilitation Unit Program Guide in 1977.

Does cardiac rehabilitation benefit the patient and, if so, which patients benefit most? Are there risks and complications involved? Is the effort justified? This article addresses these questions after a brief description of rehabilitation programs.

WHAT IS CARDIAC REHABILITATION?

Rehabilitation of coronary patients was defined by a committee of the World Health Organization in 1964 as "the sum of activity required to insure the best possible physical, mental, and social conditions, so that they may by their own efforts regain as normal as possible a place in the community and lead an active productive life".[1]

From this definition it is apparent that good medical care, although of primary importance in the rehabilitation process, will frequently need to be augmented to insure optimal rehabilitation. In addition to medical care, the major facets of rehabilitation are psychological support of the patient and his family, psychological evaluation of the patient, education of the patient and his family, counseling regarding modification of risk factors, diet, and graduated activity, assessment of work capacity, and vocational counseling. Obviously good medical care extends into many of these areas and the physician should be in full control of the rehabilitation process, but it is clear that his efforts can be enhanced considerably by several allied health professionals: cardiac rehabilitation nurse, dietician, physical therapist, social service worker, occupational therapist, clinical psychologist, and vocational rehabilitational counselor. In the out-patient exercise programs, physical education personnel are needed. Ideally, cardiac rehabilitation is provided by such a team acting at the request of the patient's physician.

Cardiac rehabilitation is usually described in three phases: In-hospital, Convalescent, and Maintenance. Detailed description of such programs can be found in the American Heart Association Cardiac Rehabilitation Program Guide.[2]

Phase I: Rehabilitation of the myocardial infarction patient begins on his admission to the Coronary Care Unit with psychological support and a positive attitude toward eventual full rehabilitation. The program is usually described in four categories: self care activities, education, exercise/physical therapy, and occupational/recreational therapy. To allow for ease of prescribing by the attending physician, the program is printed out in stages with appropriate instructions under each of the four categories for each stage.

Phase II: The convalescent phase lasts 4 to 10 weeks. During this time, the heart heals and the patient partially regains his physical fitness. Pa-

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* Medical University of South Carolina, 171 Ashley Avenue, Charleston, South Carolina 29403

tients with light or sedentary jobs return to work at the end of this phase. The main exercise is usually walking which should be prescribed by the physician in detail as to duration, frequency, and speed with appropriate advice regarding warm-up and cool-down periods and other precautions. If an organized program is available, exercise is usually supervised, monitored, and carried out on a treadmill or bicycle ergometer.

Phase III: Supervised programs are usually held in the gymnasium three times weekly and last for three to twelve months. Exercise sessions usually consist of calisthenics, walking or jogging, bicycling, games, and swimming. Exercise is prescribed by heart rate, initially 60% of maximum, progressing to 70 to 85% of maximum. Patient education and counseling regarding risk factor modification are part of the program.

If no formal program is available, the patient may be continued on a walking program, usually a maximum of three miles at a maximum speed of four miles per hour. If the patient wants to engage in more strenuous exertion, for example jogging, tennis, handball, he should undergo exercise testing first.

The exercise prescription is again based on heart rate which the patient must learn to count accurately. Usually heart rate prescriptions are lower for unsupervised exercise than for supervised.

IS CARDIAC REHABILITATION OF VALUE?

The two end points for which firm evidence is available are length of stay in the hospital with the acute event and percentage of patients returning to work. There is also some evidence for reduced rate of rehospitalization for coronary disease and a reduced mortality rate. Psychological benefits are undoubtedly real although hard to quantitate.

The average length of stay in the hospital for treatment of the acute myocardial infarction patient has been markedly reduced over the last four decades. In 1940, four to six weeks was the usual length of stay. This was shortened to three weeks by 1970[3] and presently is nearer two weeks, and even less in some centers. This is undoubtedly due to many developments but surely the concepts of rapid mobilization and active rehabilitation of the patient have played an important part. This is clearly documented by Ackers' study in a community hospital before and after introduction of a Cardiac Rehabilitation Unit. He found a four day reduction in length of stay comparing 1967-1968 admissions to those in 1969-1970.[4]

In 1940, Master and Dack[5] reported 53% of 415 patients surviving an acute myocardial infarction returning to work, about two-thirds full time. Factors in order of importance were age, the number of attacks, and occupation, professional persons being the most likely to return to work and laborers least likely. In 1971, Wiggle et al[6] reviewed the world literature on this subject and found 10 studies between 1945 and 1969. Three of these were in the United States, the most recent in 1967 and these showed re-employment rates of 72 to 74%. Other countries range from 80 to 88%.

In 1967, Seldon[7] reported his experience with the first 1,000 cases seen at the Cardiac Rehabilitation Unit in Randwick, Australia. These referrals were patients not rehabilitated by the normal process of returning to their previous job or finding a suitable alternative job. Eighty-two percent of these were considered fit for work and 75% did in fact resume work. In 1977, Degré reported 85% returning to work after a rehabilitation program versus 63% without rehabilitation. Factors of major importance were a short time interval between the myocardial infarction and beginning the rehabilitation program and the addition of psychotherapy to exercise training.[8]

Cardiac rehabilitation has been shown to be beneficial in most studies of reinfarction rates and long term survival. Haskell reviewed and tabulated nine of these studies recently.[9] Many are of questionable validity because they either had no control group or else used the drop-outs as a control group. One study by Rechnitzer[10] attempted matched controls and reported a three fold greater mortality in these patients compared to patients in exercise programs but this latter group were volunteers, hence psychological differences may play a part.

Psychological benefits, although thought to be an important aspect of cardiac rehabilitation by many investigators, are difficult to quantitate.

WHICH PATIENTS WOULD BENEFIT MOST?

While all patients benefit from the current attitudes of practitioners produced by developments in the field of cardiac rehabilitation, some patients stand to benefit most from a formal Rehabilitation Program. Examples are the anxious patient, the aggressive over-compensating patient, and those patients with the greatest likelihood of arrhythmias. The Joint Working Party on Cardiac Rehabilitation in England recommended that all patients who have not returned to work at three months after a myocardial infarction should be referred for evaluation and rehabilitation.[11]

RISKS AND COMPLICATIONS

Haskell reported data from 30 supervised programs having a total of 1,480,000 man-hours of participation. Thirty-seven non-fatal cardiac arrests or myocardial infarctions and 14 fatal events occurred in these groups for an average of one cardiac event per 29,020 man-hours.[9] Unsupervised activity obviously carries a greater risk and places more responsibility on the physician prescribing the exercise.

CONCLUSION

The concepts of rapid mobilization of the patient after a myocardial infarction and a team approach to full rehabilitation of the cardiac patient have gained widespread acceptance in the past two decades. Formal rehabilitation programs, both inhospital and outpatient supervised conditioning programs, are being set up in many centers utilizing several allied health professionals in the rehabilitation effort. There is some firm evidence that this has resulted in a decreased length of stay in the hospital and an increase in the percentage of patients returning to work. The evidence for decreased reinfarction rate and mortality rate is less reliable, and the psychological benefits, although conceded to be real, are difficult to quantitate.

Patients who benefit most from a formal outpatient supervised program are those who have not returned to work by three months after their myocardial infarction.

The present evidence of benefit from Cardiac Rehabilitation Programs is sufficient to justify the effort and expense incurred in establishing such programs and one is being established now at the Medical University of South Carolina. □

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Editorials

ON SYMPOSIA, CASE REPORTS, THE ENERGY CRISIS AND "THROWAWAY" PERIODICALS

This issue of *The Journal* features a symposium on current perspectives in cardiology, edited by Doctors Usher and Lizarralde of the Medical University of South Carolina. A survey of our readership, taken at the SCMA Convention in 1976, indicated an interest in "concise, practical review articles"; certainly, all of the articles edited by Doctors Usher and Lizarralde fit in this category.

While we will continue to strive for symposium issues of *The Journal* from time to time, it seems worthwhile to emphasize again that *The Journal* exists, in large measure, as a place for practicing physicians to publish their observations. *Case reports remain welcomed!*

The Journal of the South Carolina Medical Association was founded, in 1905, by practicing physicians, for practicing physicians. At that time, the state had no full time faculty. With the growth of full time faculties, there has been a trend for practicing physicians to do less and less writing, which seems in some ways unfortunate.

In the corridors of our hospitals, one frequently overhears the remark, "We ought to write this one up!" We are aware of no published observations on the statistics of what happens to those desires to "write this one up." We would suspect, based on casual, uncontrolled observations, that the statistics would be more or less as follows: of every 1,000 times that someone states "We ought to write this one up," there may be 100 trips to the library, ten efforts to start a manuscript, and one finished manuscript sent in for publication. This sort of selection process is valuable in "keeping down" the deluge of literature with which physicians are confronted. Nevertheless, one wonders what we have missed.

It remains our feeling that case reports are extremely valuable means of self-expression by practicing physicians. Even if the case adds "nothing new," a concise review of the literature is often beneficial to readers. And, of course, the greatest gain goes to the author for the self-discipline involved in preparing and organizing the manuscript.

This leads us to the next topic — the energy crisis. Our local Columbia newspaper has been making quite an issue, recently, of ways to cut down on energy by delivering the mail more efficiently. It has been suggested several times that door-to-door delivery be discontinued. Rather, everyone should have a box at the curbside, in which the carrier can place mail, thereby saving fuel.

Sadly, I do not think that this exemplary suggestion would be applicable to those of us in the medical profession, unless we substituted the curbside "box" for a *bin!* The problem of "throwaway" journals and other unsolicited periodicals in mail delivered to physicians is simply overwhelming. I have often lamented the number of trees which seem to be felled in my behalf annually, and now I share the concerns of others about the effect of all of this mail on our supply of fuel.

"Throwaway" periodicals often have helpful information, but we feel that some re-evaluation of the role of these periodicals in our lives is perhaps in order. The results of their readership surveys have, to our knowledge, not been published. One wonders what percentage of the actual scientific content is in fact read. It remains our feeling that the incredible number of "throwaway" periodicals is overwhelming to the recipients, who might be better served by focussing their attention on only a few, preferred periodicals run *by and for* the medical profession itself.

— CSB

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President's Page

THE AMERICAN MEDICAL ASSOCIATION

A detailed report of the recent AMA meeting in Chicago will be made to you by the elected delegates, but since I want to brag a bit about them, they probably will forgive me for preempting them.

I am sure that you would be proud of the effectiveness of your current South Carolina Delegation to the AMA. Included in this Delegation are the Auxiliary and members of the Housestaff Physicians, in addition to the regular delegates. They all worked hard to make your voice and your opinion heard at the national level, and they worked together.

Dr. John C. Hawk, Jr. was elected on the first ballot, from a field of six, to one of the two contested slots on the Council on Constitution and Bylaws. He will serve in his usual efficient manner, and will be a credit to our state.

Mrs. Wayne C. Brady of Greenville was re-elected to the Board of Directors of the AMA Auxiliary. Mrs. Brady has served the Auxiliary with dedication in many state positions. She now is in a position of responsibility and honor which she justly deserves. Many other Auxiliary members from the state attended, and from the bits of conversation I have heard from them, politics and legislative persuasion are not foreign subjects to them. Service is their motto, and we should never forget the effort that is put forth by the Auxiliary in behalf of the AMA-ERF. Their annual contribution exceeds one million dollars. I am pleased at the interest expressed by the Auxiliary officers in behalf of "*Better Health Through Health Education.*" We will certainly hear more from them.

Dr. Rick Richards, a family practice resident on the house staff at Spartanburg General Hospital, was a principal in a morning-long program given on "*Lifestyles and Their Relation to Health*" during the AMA meeting. Many favorable comments were made following this program. It is encouraging to see the dedication, determination and devotion to a cause or belief demonstrated by many young physicians. Housestaff membership in the AMA and SCMA is growing. We welcome their participation. Rick Richards did his part to make South Carolina visible.

Several issues seemed to stand out during this session, two of which are: First, *National Health Insurance*. The House reaffirmed its confidence in the Board of Trustees to handle this issue using the guildelines established in Resolution 62. This Resolution, made by the Florida Delegation at the last meeting, calls upon the AMA to oppose any and all federally controlled compulsory health insurance programs, and directs that continued support be given to the private health insurance industry for extension of coverage. The second, the much debated issue of changes being proposed to the *Principles of Medical Ethics*, is unresolved. The amended version will be distributed to state associations, component medical societies and specialty societies for study prior to the Interim Meeting in 1979. The final report will be presented at the Annual Meeting in 1980. I hope that you will take the time to review this document when it is presented to your society.

Again, I am proud and pleased with the activities of those who attended the AMA meeting, and hope that more of you will make an effort to attend future meetings.

HARRISON L. PEEPLES, M.D.,
President

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Watch for these proceedings in the November, 1979 issue of The Journal

SOUTH CAROLINA MEDICAL ASSOCIATION

AUXILIARY

AMA-ERF

The South Carolina Medical Association Auxiliary is fortunate to have two energetic and creative ladies serving as Chairman and Co-Chairman of AMA-ERF for next year.

Serving as Chairman will be Carol (Mrs. Newman) Harter of Anderson. Her husband, Newman, is a Family Physician and they are the parents of two children, Kendall and Tradd. For the past two years, Carol has done an outstanding job as Chairman of AMA-ERF for the Anderson County Medical Association. She directed the sale of hand-painted eyeglass lenses which proved to be a profitable effort for the Auxiliary.

Jean (Mrs. DeWitt) Niles is serving as Co-Chairman for the coming year. Also from Anderson, Jean and her husband, Dr. DeWitt Niles, an Anesthesiologist, are the parents of three children, Paige, Bradley, and Russell. Jean has served in many capacities with the Anderson County Medical Auxiliary with the most recent being that of President. Jean initiated the Cancer Screening Clinic which has become an annual project.

Several Auxiliaries in the state have found various projects which have turned into money making efforts for the American Medical Association Education and Research Fund.

In Spartanburg, the Auxiliary found that they are their own best customers when they held a talent auction. Mrs. Brownlee Lowry served as Auctioneer for the affair which brought in a net result of $1,114.00 The upcoming Chairman for this year will be Mrs. Woodliff Sanford.

The Columbia Medical Auxiliary has used the Christmas Sharing card for the past few years and has found it to be very successful. The front of the Christmas card is designed by an Auxiliary member. The Auxiliary raised over $3,000.00 last year with this card. Co-Chairmen for this year are Mrs. Jeff Brooker and Mrs. Dan Paysinger. Designing the front for the Christmas card will be Mrs. Rod Macdonald.

The past year has been a busy one for the Sumter-Clarendon Auxiliary. They sold note cards, calendars, watches and held an auction. Their plans for a 1980 project are underwas with Mrs. G. Murrell Smith serving as Chairman of AMA-ERF.

Mrs. Newman Harter
AMA-ERF Chairman

Mrs. DeWitt Niles
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THE JOURNAL
OF THE SOUTH CAROLINA MEDICAL ASSOCIATION

VOLUME 75 OCTOBER 1979 NUMBER 10

THE LASER — DEATH RAY?? LIFE RAY!!

JON I. SATTLER, M.D.*

The word "laser" conjures up images of star wars, robots, and surrealistic landscape. Since the first operational laser was built by Dr. Maiman in 1960, the laser has instigated more fear, excitement and imagination than any other recent scientific technology. The growth of laser technology is probably unparalleled by any other phase of scientific growth in our history.[8]

First of all, what is a laser? L.A.S.E.R. is an acronym representing *Light Amplification* by *Stimulated Emission* of *Radiation*. What does that mean? Basically, radiation is electromagnetic energy which has a spectrum ranging from sound to penetrating radiation, e.g. x-ray. The middle of this spectrum is light, in the visible spectrum of energy. The term Light Amplification is the increasing of energy output of visible light. To have an output of energy, you must have a mechanical means of increasing the output of light. Stimulated Emission of light begins in a resonating chamber. This light then increases its energy level by resonating, (Amplification), until the force of energy exceeds the capacity of the chamber to retain the energy. This released energy is concentrated light energy.[8]

The light described above is called "coherent" light. How does this differ from the light from the sun or a light bulb? The latter are incoherent light, meaning their light energy is scattered over a wide area. Coherent radiation is, however, focused in a high intensity beam.

The development of the laser has opened the door to the new technology of photonics. Photonics, literally the science of light, gives us the electronics of the twenty-first century. Now that this energy with its intensity of beam and almost limitless temperatures has been defined and controlled, what do we do with it?[3]

The impact of laser on the commercial world has forced the definition of accuracy to be rewritten. Consider accuracy in measurement to one millionth of a centimeter. It is this accuracy which has allowed man to walk on the moon. This accuracy can measure the difference in distance between the earth and the moon to within one foot. Lasers are used to measure the speed of light and in the measurement of hydrogen frequency.[3]

Commercially, the laser has influenced technology from communication in its most sophisticated form to the surveying of land. In communication, the laser is used to produce audiovisual reproductions of pictures and sounds. The end product is referred to as "laser hi-fi." Three dimensional pictures known as holograms are produced and reproduced by laser. These holograms appear almost as miniature motion pictures. The laser phone cable can carry more information than a conventional electrical telephone cable.[3]

The laser has revolutionized the world of journalism. The rapidity of transmission possible through the laser enables you to watch an event as it happens on the other side of the globe. The Los Angeles Times uses lasers to transmit written news bulletins from one area to another.[3]

Monitoring vulnerable stress points accurately by laser offers a quality control and safety feature for the world of construction. The laser is also used to test welding for precision and to harden metal surfaces. The laser has the ability to form new metal alloys directly on a surface. The ability of the laser to package and deliver a large amount of energy in a very small area makes it a machine designed for accuracy.[3]

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* 3321 Medical Park Road, Columbia, S. C. 29203

In our own immediate economic environment, textiles, the laser can be used to cut fifty suits simultaneously. With no frayed edges on the cloth! Maneuverability makes the laser a most efficient pair of scissors.[3]

The adventures of James Bond have been hitting close to home. Scientific and military utilization of the laser has brought Dr. No's attempt to do in 007 to the kindergarten category in comparison with 100% accuracy destruction devices for long-range projectiles.[3]

The laser can be used to freeze a molecular event as it takes place. The implications in research of this one ability is mind-boggling. "Shiva," a giant laser capable of producing forty trillion (trillion, that is not a misprint) watts, is used in nuclear fusion research. Temperatures of up to one hundred million degrees Fahrenheit can be produced by a laser.[3]

What has the military been up to? If you have listened to the news lately or read any discussions of SALT II you will be familiar with the "Death Ray" device that the Russians have allegedly built. This area of military armament has been played down because it is extremely controversial and has an immediate reaction of terror. A few of the current weaponry uses of lasers are listed below.[3]

The military use of lasers in gunnery range-finding has an accuracy level that is classified. In the infantry, lasers are used for target designation. Air craft can hone in on specific laser lengths and discharge laser-seeking missiles. Other uses of lasers in aircraft are classified, as are most of the uses for tanks. We do know that lasers are used on tanks for range-finding and target designations. We do not know if the laser is used as an actual weapon. Yet![3]

Military spokesmen have been quoted as saying that lasers are easily maneuverable and result in a rapid rate of "kill." Power ranges have been listed as high as 100,000 kilowatts.[3]

How can we turn this potential "Death Ray" into a real and valuable "Life Ray"?

In 1969, Dr. Leon Goldman began to use the laser in eye surgery. Currently laser treatment (photocoagulation) is used for treatment of progressive diabetic retinopathy, hemorrhage, central serous macular edema, retinal detachment and so-called histoplasmosis of the eye.[4] In 1977, Gerald Stiegler published a paper on the new glaucoma research laser. This paper discusses procedures for laser surgery such as iridectomy, iridotomy, pupil plasty and trabeculotomy.[10]

About the same time Goldman began ophthalmological use of the laser, Dr. Harvey Lash, a California plastic surgeon began to treat lesions of the face with laser energy. His associate, Dr. David Apfelberg reports good to excellent results in treating cutaneous vascular abnormalities with the argon laser.[1, 2] Research has also proved the laser useful in the treatment of burns, stimulating wound healing and skin-grafting.[9]

Hepatic and gastric hemorrhages have been rapidly controlled by laser. As a matter of fact, in comparing blood loss in cold-scalpel incision to blood loss in laser incision, the blood loss was minimal in laser use.[4, 6]

CO_2 lasers have been also used to treat lesions of the vocal cords and to vaporize tumors of the cervix. As you may guess, the list of uses for lasers is growing by the day.[4]

The argon laser was developed as an exciting advance of earlier lasers and is used in the treatment of port wine hemangiomas, capillary/cavernous hemangiomas and telangectasia, nevi aranei, tattoos, moles, and various pigmented lesions. These different lesions are at least now treatable when previous treatment was at best poor.[5]

In Columbia, we now have an argon laser at the Laser Institute. This is being used to treat the above named lesions and to conduct clinical research; currently in the area of vascular lesions of the lower extremities, e.g., spider and burst veins.

The institute has plans for improving the current use of argon for skin lesions and to move into a more complex surgical research involving CO_2 for wound debridement, and perhaps Yag-Nd lasers for another approach to pigmented lesions.

The purpose of this paper is to discuss the laser, its industrial and medical uses. The "Life Ray" opens doors to previously unexplored corridors of medical treatment and gives rise to infinite possibilities to improve the quality of life. ☐

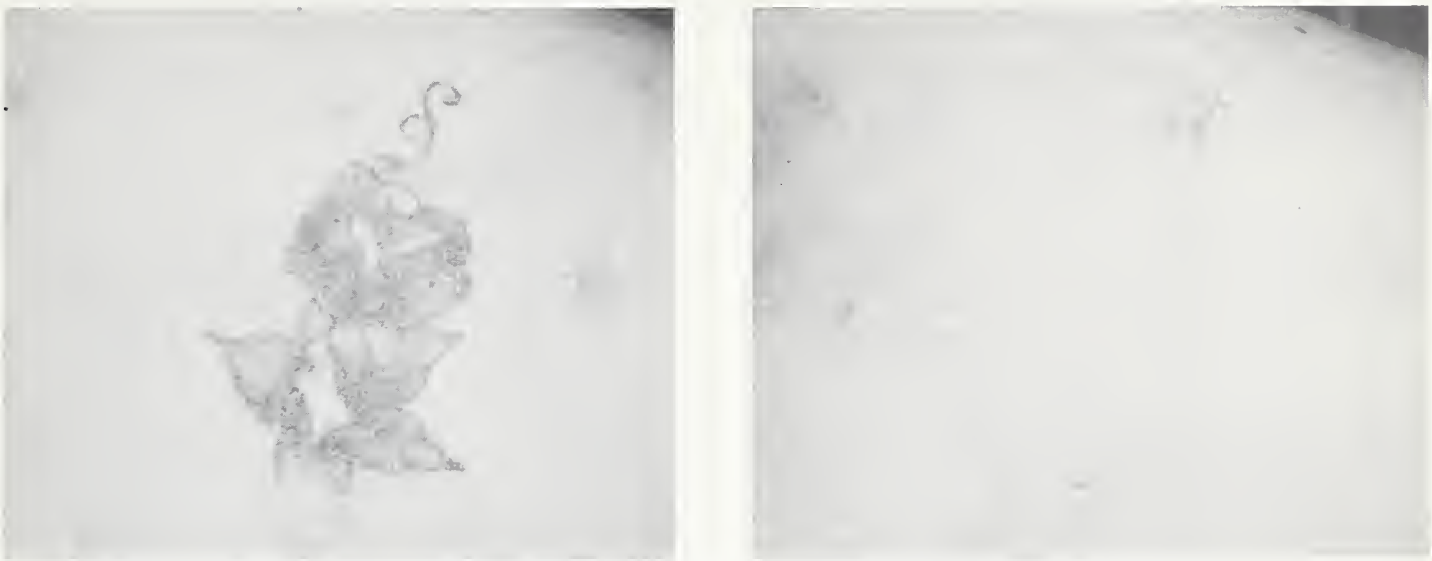

FIGURE 1. Tattoo of arm before and after argon laser therapy.

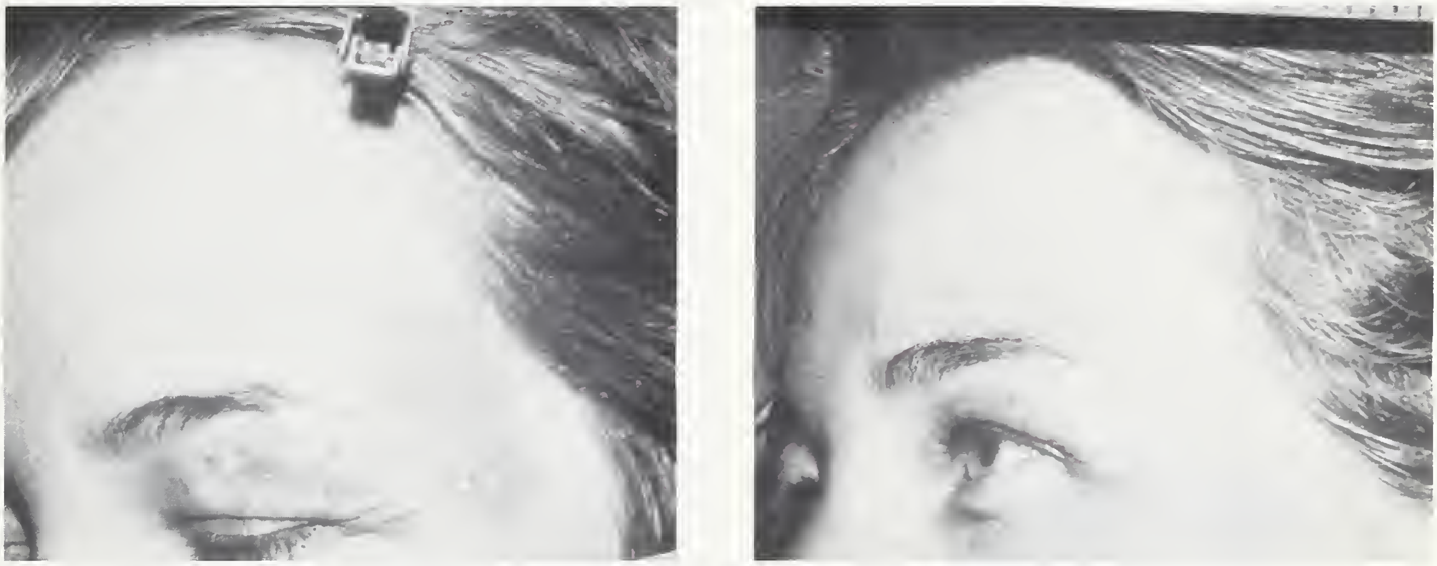

FIGURE 2. Port wine stain before and after argon laser therapy.

ACKNOWLEDGEMENT

I would like to express my sincere appreciation to Nancy M. Turner, R.N., for her assistance in collecting, collating and editorializing, without whom this publication would never have been written.

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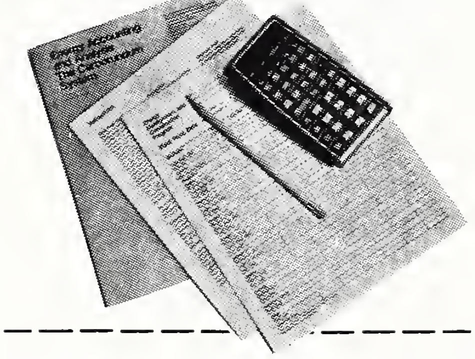

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WARNINGS: If tolerance develops, the recommended dose should not be exceeded in an attempt to increase the effect; rather, the drug should be discontinued. Tenuate may impair the ability of the patient to engage in potentially hazardous activities such as operating machinery or driving a motor vehicle; the patient should therefore be cautioned accordingly. *Drug Dependence:* Tenuate has some chemical and pharmacologic similarities to the amphetamines and other related stimulant drugs that have been extensively abused. There have been reports of subjects becoming psychologically dependent on diethylpropion. The possibility of abuse should be kept in mind when evaluating the desirability of including a drug as part of a weight reduction program. Abuse of amphetamines and related drugs may be associated with varying degrees of psychologic dependence and social dysfunction which, in the case of certain drugs, may be severe. There are reports of patients who have increased the dosage to many times that recommended. Abrupt cessation following prolonged high dosage administration results in extreme fatigue and mental depression; changes are also noted on the sleep EEG. Manifestations of chronic intoxication with anorectic drugs include severe dermatoses, marked insomnia, irritability, hyperactivity, and personality changes. The most severe manifestation of chronic intoxications is psychosis, often clinically indistinguishable from schizophrenia. *Use in Pregnancy:* Although rat and human reproductive studies have not indicated adverse effects, the use of Tenuate by women who are pregnant or may become pregnant requires that the potential benefits be weighed against the potential risks. *Use in Children:* Tenuate is not recommended for use in children under 12 years of age.

PRECAUTIONS: Caution is to be exercised in prescribing Tenuate for patients with hypertension or with symptomatic cardiovascular disease, including arrhythmias. Tenuate should not be administered to patients with severe hypertension. Insulin requirements in diabetes mellitus may be altered in association with the use of Tenuate and the concomitant dietary regimen. Tenuate may decrease the hypotensive effect of guanethidine. The least amount feasible should be prescribed or dispensed at one time in order to minimize the possibility of overdosage. Reports suggest that Tenuate may increase convulsions in some epileptics. Therefore, epileptics receiving Tenuate should be carefully monitored. Titration of dose or discontinuance of Tenuate may be necessary.

ADVERSE REACTIONS: *Cardiovascular:* Palpitation, tachycardia, elevation of blood pressure, precordial pain, arrhythmia. One published report described T-wave changes in the ECG of a healthy young male after ingestion of diethylpropion hydrochloride. *Central Nervous System:* Overstimulation, nervousness, restlessness, dizziness, jitteriness, insomnia, anxiety, euphoria, depression, dysphoria, tremor, dyskinesia, mydriasis, drowsiness, malaise, headache; rarely psychotic episodes at recommended doses. In a few epileptics an increase in convulsive episodes has been reported. *Gastrointestinal:* Dryness of the mouth, unpleasant taste, nausea, vomiting, abdominal discomfort, diarrhea, constipation, other gastrointestinal disturbances. *Allergic:* Urticaria, rash, ecchymosis, erythema. *Endocrine:* Impotence, changes in libido, gynecomastia, menstrual upset. *Hematopoietic System:* Bone marrow depression, agranulocytosis, leukopenia. *Miscellaneous:* A variety of miscellaneous adverse reactions has been reported by physicians. These include complaints such as dyspnea, hair loss, muscle pain, dysuria, increased sweating, and polyuria.

DOSAGE AND ADMINISTRATION: Tenuate (diethylpropion hydrochloride): One 25 mg. tablet three times daily, one hour before meals, and in midevening if desired to overcome night hunger. Tenuate Dospan (diethylpropion hydrochloride) controlled-release: One 75 mg. tablet daily, swallowed whole, in midmorning. Tenuate is not recommended for use in children under 12 years of age.

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PULMONARY RESECTION IN METASTATIC CANCER OF THE LUNG*

L. DIETER VOEGELE, M.D.**
ABD-EL-AZIZ RICHI, M.D.
WILLIAM H. PRIOLEAU, JR., M.D.
PETER HAIRSTON, M.D.

The appearance of metastatic lesions on a chest x-ray in a patient with a primary cancer elsewhere need not indicate disease uncontrollable by surgical treatment. It is unfortunate that many clinicians view this aspect with helplessness since surgical removal of pulmonary metastases can result in prolonged survival. Among fatal cases of malignant disease Willis identified 30% to have lung metastases, half of whom had lung deposits only.[1, 2]

Resection of such lesions was performed as early as 1898[3, 4]; however, the real concept of operative treatment for cure received its impetus from the 1939 report by Barney and Churchill[5] of a pulmonary resection for metastasis in a patient with adenocarcinoma of the kidney. The patient eventually survived 23 years.

In subsequent years, surgeons advocated resection only in patients with solitary metastases, confined to one lung, after a prolonged disease-free interval. A 1947 review listed approximately 24 cases.[6] By 1957 an additional 300 cases had been published.

HISTOLOGICAL TYPES

Osteogenic sarcoma and mesenchymal tumors frequently metastasize to the lungs first.

Studies by Martini[8] had shown that in patients with sarcoma 83% of them developed metastases within two years and 95% were dead within three years without any five year survivors. However, patients who underwent resection of pulmonary metastases had a 45% three year survival. Other sarcomas (except lymphoid types) had similar results.

Testicular tumors (germinal cell origin) had a survival of 31% at five years treated by combined surgery and adjuvant therapy. Metastatic lesions in the chest following head and neck carcinoma had a 44% five year survival following resection.[9] Figure 1 shows cumulative survivals in resected metastases in a series of patients, not including sarcoma patients, compared to a similar group of nonoperated patients.

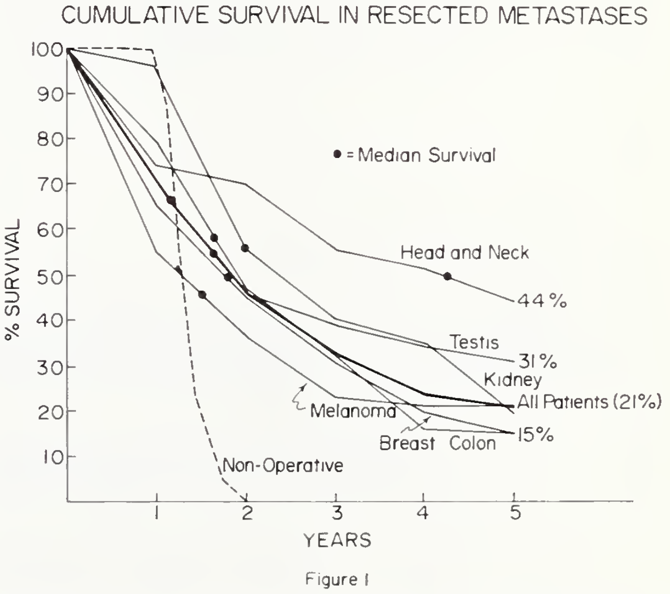

FIGURE 1. Cumulative survival in resected pulmonary metastases.

INTERVAL

Timing of surgical intervention is controversial. Biologic behavior of the malignancies as well as host tumor relationships are variable. Most reports emphasize that tumor-free interval seldom affects survival from the metastatic disease in the lungs.[10, 11, 12] Even patients requiring secondary resections for pulmonary recurrences have an appreciable five year survival.[10] Nevertheless, other workers felt that there existed a correlation between tumor rate of growth and disease-free interval. Collins,[7] through work done in animals and in clinical work, felt that tumors grew at a predictable rate and that tumor doubling time defined tumor behavior. In some instances correlations could be established between tumor doubling time and disease-free intervals.[13]

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EXTENT OF SURGERY AND APPROACH

In the past surgeons advocated pulmonary resection only in patients with solitary metastases confined to one lung after a long disease-free interval. However, more recently, the approach has been to search for the asymptomatic patient as this seems to carry the prognostic marker.[11] Prognosis has been seen to be quite unaffected by:

1) Extent of disease;
2) Disease-free interval;
3) Tumor doubling time.

As can be seen from Table 1 and Figure 1 breast, melanoma, and colon are most frequently single metastases yet have universally the lowest five year survival following resection of metastases. On the other hand, head and neck tumors have the fewest single metastases yet have the best five year survival following resection. Particularly in metastatic sarcomas, multiple lesions can still be associated with improved survival.[12] Unilateral and bilateral disease is amenable to resection as well.[14] In a study by Wilkins[10] there was no difference in survival among patients who had a disease-free interval shorter than two years as opposed to patients with an interval greater than two years. Great variability of tumor doubling time for specific tumors has hampered the usefulness of this technique. Several authors have not found it useful to correlate with prolonged survival.[10, 11, 12]

EXTENT OF PULMONARY METASTASES AT INITIAL THORACOTOMY

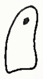
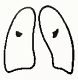
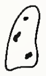
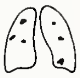
PRIMARY SITE					NUMBER OF PATIENTS
Colon and Rectum	23	1	16	6	40
Melanoma	23	0	5	1	29
Breast	24	0	3	1	28
Testis	8	0	14	3	25
Head and Neck	10	0	12	1	23
Kidney	6	1	4	5	16
Bladder	2	0	4	2	8
Other	8	0	6	5	19
TOTAL	104	2	58	24	188

Table I

TABLE 1. Extent of pulmonary metastases at initial thoracotomy.

Some factors, however, have been implicated with adverse prognosis:

1) the symptomatic patient;
2) site of origin of primary tumor (melanoma, breast, etc.);
3) lymph nodes in the lung specimen (The status of the lymph nodes at the primary resection does not seem to influence the prognosis as much);
4) pneumonectomy in order to control the metastatic disease.

In evaluating patients with carcinoma and pulmonary metastases several considerations must be taken into account. Again, it is primarily the asymptomatic patient who will be amenable to resection therapy. Extrathoracic spread must be ruled out. Patients with a history of cancer and lung lesions should be diligently evaluated. Occasionally a benign tumor is found.[15]

The surgical approach is predicated upon:

1) conservation of lung tissue;
2) removal of all visible disease.

These two objectives can fortunately frequently be met because metastatic spread in the lungs usually occupies a subpleural position. Thus in 70%, wedge resections can be accomplished. Approximately 20% lobectomies are required and rarely will a pneumonectomy need to be relied upon to remove all visible disease.

Single lesions are approached by standard posterolateral thoracotomy. Bilateral lesions can be staged or treated preferably via a median sternotomy. This is a favorable approach because it allows inspection of all lung surfaces and is associated with slightly decreased post-operative pain and ventilatory dysfunction.[14] The only drawback is the relatively difficult access to the left lower lobe because of the position of the heart.

SUMMARY

Metastatic lesions in the lungs may be amenable to surgical therapy. Prognosis has been unaffected by extent of disease, disease-free interval or tumor doubling time. Solitary and multiple lesions when resected can yield impressive survival figures. The future holds promise with adjuvant chemotherapy. The operative principles for surgical therapy of metastatic disease to the lung are:

1) Primary tumor must be controlled or controllable (by resection);

2) No extrathoracic disease;
3) Good operative risk;
4) No available effective therapy solely by non-surgical means. ☐

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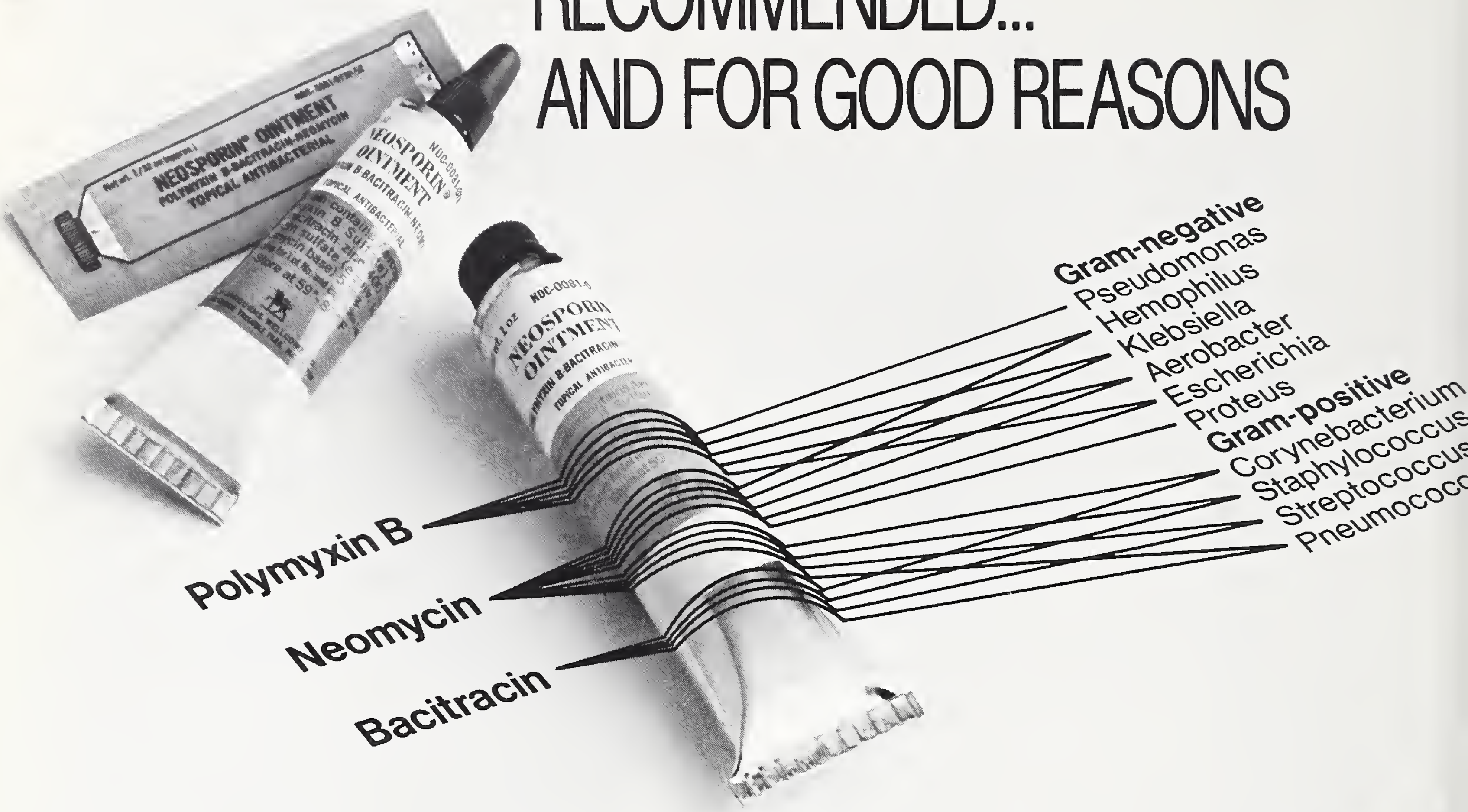

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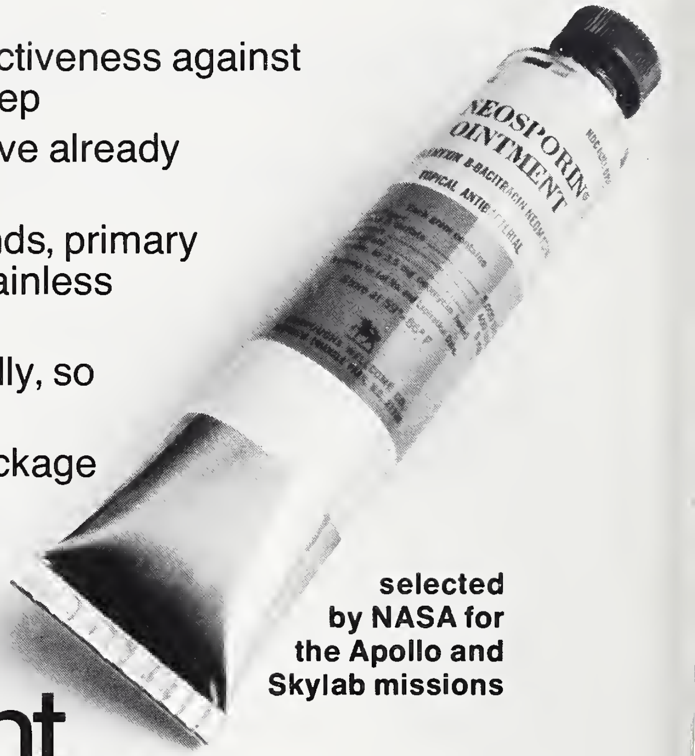

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INSECT ALLERGY REVISITED

CHARLES H. BANOV, M.D., F.A.C.P.*

Since it is the responsibility of consultants in our state to keep all of the physicians up to date on new advances in their fields, I felt it necessary to review some of the new information about insect stings. Most physicians are now aware of the potential seriousness of stinging insect allergy. Our state has shared some of the 40 fatalities which occur each year as a result of insect stings. I have written in this journal in the past warnings that perhaps many undiagnosed fatalities in our state could well have been due to stinging insect allergy. The coronary or stroke which occurs in a young individual suddenly might well be a simple sting of a bee, wasp, yellow jacket or hornet. The frustrating aspect of this is that THESE TRAGEDIES COULD WELL HAVE BEEN PREVENTED BY AVAILABLE TREATMENT!!! The treatment up to now has been fairly effective but the newly developed venom therapy is even more effective and protective.

For many years, allergists have had to rely on whole body extract for diagnosis and treatment. Some studies done through the American Academy of Allergy have certainly indicated some degree of effectiveness from this material. However, recent research, primarily that of Dr. Lichtenstein at Johns Hopkins and Dr. Robert E. Reisman in Buffalo has indicated that the venom of the stinging insect is probably the agent producing the anaphylactic reactions. Using some of the new immunodiagnostic methods, it has been shown that those patients treated with insect venom developed laboratory as well as clinical evidence of protection after treatment.

Venom materials are so specific that in a truly insect allergic individual, there is a significant possibility of untoward allergic reaction during skin testing. This reaction can vary from mild urticaria to a life threatening laryngeal edema or bronchospastic episode. Skin testing should only be done by those well experienced with allergic reactions and in fully equipped facilities for treating life threatening anaphylaxis. Patients should be fully informed of the intrinsic dangers of skin testing before it is done and the patient should sign a statement that he has been so informed for legal protection of the physician.

Furthermore, skin testing is done with the actual venom materials. These are expensive. Although one diagnostic testing kit can last for an entire month, some of the dilutions for testing must be made up freshly the very day of the skin testing. Some dilutions can be made less frequently.

In interpreting the results of skin testing, the clinical story must be carefully evaluated. Many patients will have non-specific positive reactions to the venom materials. This is less likely to occur at the weaker dilutions and the recommendation has been made that if patients react to a dilution of 1 mcg or less that treatment should be strongly considered if there is a significant history of prior constitutional reactions to stinging insects.

Although patients must be tested with individual venom, there is some cross reactivity among the stinging insects. The vespids (yellow jacket, hornet and wasp) do tend to cross react but generally do not with the honeybee which appears to be a distinct hymenoptera. If one is sensitive to the bee, he can receive that particular material and if there is reaction to the wasp,

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yellow jacket or hornet, a combination of those materials could be given.

Unfortunately, the supply of venom materials in the world is still less than demand. In the United States, only one laboratory is licensed to prepare this material. All patients who are currently on whole body extract should be eventually brought back to the physician and retested with the new venom materials. However, this cannot possibly be done all at once as there is simply not enough venom to meet the needs. Accordingly, as patients come in for re-evaluation and as new patients are started on treatment, the venom materials should be used. For those patients who are currently on maintenance dosages of whole body extract, temporarily, it is recommended that they be maintained status quo on their material until re-evaluation can be done. There is no way to convert an individual on whole body extract to venom therapy. They are simply two distinctly different forms of therapy.

The following summary of the current status of stinging insect treatment is as follows:

(1) All patients who have had a true constitutional reaction to stinging hymenoptera (bee, wasp, yellow jacket or hornet) should be considered for immunotherapy with the new venom materials. Appropriate diagnostic studies such as skin testing and/or invitro (radioallergoabsorptive studies) should be done prior to the institution of immunotherapy.

(2) As soon as a history of insect hypersensitivity is made, the patient should be provided with an Epinephrine syringe and instructions for emergency use should a repeat sting occur prior to the time that definitive therapy can be instituted.

(3) No new patients should be started on whole body extract.

(4) All patients should agree to have immunotherapy with the venom materials instituted prior to having skin tests with the new venom materials. There would be no logic in doing skin testing and making a definitive diagnosis of insect allergy if one were not then to proceed with appropriate therapy. This would be unnecessary jeopardy to the patient and certainly unnecessary expense.

(5) All physicians dealing with either diagnosis or treatment of venom sensitive insect hypersensitivity cases should be fully prepared for immediate and prolonged treatment of anaphylactic reactions.

(6) Venom therapy with the stinging insects has proven to be extremely effective for the 0.4% of the general population who may be affected by severe allergic reactions to stinging insects. The anticipated over 90% effectiveness of this therapy should allay the fears of the patient and physician of potentially fatal reactions from stinging insects. ☐

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CURRENT DIAGNOSIS AND TREATMENT OF ACUTE ANAPHYLAXIS

TIMOTHY A. LAMPHIER, M.D.*

Anaphylactic reactions are the immediate shock-like and frequently fatal reactions which may occur within minutes after exposure to various antigens. There are three major classes of offending antigens: *proteins*, *polysaccharides*, and *haptens*. *Haptens* are partial antigens which become immunogenic when they or their metabolites form stable bonds with carrier proteins to form complete antigens. The antigens of many drugs and diagnostic agents fall into this last category.

A large number of antigens may precipitate anaphylaxis; these are summarized in Table I. These antigens are capable of stimulating the production of immunoglobulin E (IgE). An individual becomes sensitive to a particular antigen after producing IgE to this antigen. A true allergic reaction is one which is immunologically mediated — that is, mediated by an antibody following re-exposure to an antigen.

IgE is produced by plasma cells in lymph nodes located primarily in the oropharyngeal area, bronchial mucosa, and intestinal lymph nodes. This humoral immunoglobulin contains the *reaginic antibody* that plays a primary role in mediating many hypersensitivity type allergic reactions. This immunoglobulin attaches to mast cells and basophilic leukocytes. When the cell-attached IgE antibody combines with an antigen, the resulting complex disrupts the mast cell or basophil membranes.

These cells contain histamine and a precursor substance for slow reacting substance of anaphylaxis (SRS-A). When an antigen combines with the IgE on these cells, there is subsequent release of the mediators of anaphylaxis: histamine, SRS-A, eosinophilic chemotactic factor of anaphylaxis (ECF-A), kinins, and prostaglandins. These chemical mediators act on shock organs, such as the bronchioles and the skin. These mediators will travel to receptors in various target organs and exert their pharmacologic effects. The organs primarily affected are the skin, respiratory tract, gastrointestinal tract, and cardiovascular system.

Sensitization may follow the first exposure to the antigen or only after numerous encounters.

Eosinophilia and increased serum IgE levels are characteristic of but not pathognomonic for atopic disorders. The serum IgE levels do not necessarily parallel the clinical degree of the allergic response.

TABLE I

MAJOR CAUSES OF ANAPHYLAXIS

ANTIBIOTICS
    Penicillins; Cephalosporins; Chloramphenicol; Colistin; Aminoglycosides; Tetracyclines; Others

BIOLOGICALS
    Foreign Serums; Chyotrypsin; Gamma Globulin; Asparaginase; Polypeptide Hormones; Influenza Vaccine; Tetanus Toxoid; Measles and other egg-based vaccines

INJECTABLE MEDICATIONS
    Iron Dextran (Imferon); Dextran; Methylergonovaine Maleate (Methergine); Nitrofurantoin

LOCAL ANESTHETICS

ASPIRIN (acetylsalicylic acid)

DIAGNOSTIC AGENTS
    Iodinated contrast media; Sulfobromphthalein (BSP)

HYMENOPTERA STINGS (Bee, yellow-jacket, wasp, hornet)

ALLERGIC EXTRACTS (skin testing and treatment solutions)

FOODS (especially eggs, nuts, cottonseed, shellfish)

INTRAVENOUS NARCOTICS (Heroin)

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The radioallergosorbent test (RAST) is an "in vitro" method of measuring allergic-specific IgE in patients in whom intradermal testing cannot be used.

In anaphylaxis, there is a massive release into the cardiovascular system of allergic mediator substances, e.g., histamine, slow-reactive substance of anaphylaxis (SRS-A), kinins, and anaphylatoxins. The result is generalized vasodilation, increased vascular permeability, urticaria, bronchospasm, and glottic and subglottic edema. Bradykinin is considered to be responsible for the severe hypotension.

The three major clinical patterns of anaphylaxis are:

(1) Urticaria and angioedema.
(2) Respiratory distress caused by laryngeal edema and/or severe bronchospasm.
(3) Severe hypotension.

Table II provides a flow sheet which may be helpful in the evaluation of the patient with anaphylaxis, on first encounter in the emergency room or elsewhere. The exact date and time should be recorded. Outlined in Table II are the many symptoms and physical findings of anaphylaxis. Awareness of the protean symptoms may facilitate early recognition of an impending crisis.

Table III provides a flow sheet which may be helpful in the management of the patient with anaphylaxis. The components of this flow sheet can be described more fully as follows:

A. *Airway*
  1. Place patient flat on back.
  2. Hyperextend head and neck.
  3. Plastic airway tube and oxygen.
  4. Suction.
  5. Keep mouth open and pull tongue forward.
  6. Ventilatory assistor apparatus if needed.
  7. ???Bronchoscopy to remove secretions.
  8. Perform endotracheal intubation with assisted respiration if effective oxygenation cannot be established in one to three minutes or if there is obstruction due to bronchospasm or upper respiration obstruction (laryngeal edema).
  9. Perform tracheostomy if oxygenation cannot be sustained.

TABLE II

ANAPHYLAXIS
FLOW SHEET

SYMPTOMS AND PHYSICAL EXAMINATION

Date — Time
Labored Breathing
Malaise
Anxiety
Pruritis
Generalized Edema
Difficulty Swallowing
Hoarseness
Thickened Speech
Apprehension — Impending Disaster
Dyspnea
Glottic or Subglottic Edema
Diarrhea
Nausea
Vomiting
Faintness
Choking
Cough
Dilation of Pupils
Urticaria
Erythema — Vasodilation
Wheezing — Bronchospasm
Tachycardia
Angioedema
Itching
Constriction of Chest
Dizzyness
Weakness
Confusion
Cyanosis
Incontinence
Fever
Headache
Polyarthritis
Hypotension
Coma

B. *Adrenalin*
   1. Inject 0.5-1.0 cc (1:1000) into BASE of tongue.
   2. Subsequent injections I.V. or subcutaneously every 15-20 minutes.
   3. Inject Adrenalin into puncture site of offending agent.
   4. Dilute 1.0 cc of 1:1000 in 10 cc of physiologic saline and give over 5 minute period.
   5. 0.5 cc to be given locally at site of insect sting.

C. *Shock*
   1. 15-30 mg. Wyamine I.V. push.
   2. Levophed 2-3 ampules, plus Regitine in equal ampules, in one liter D5W, give I.V. drip.
   3. Foley catheter — keep hourly urine output over 40 cc/hour.

D. *Sedation*
   1. Seconal 100-300 mg. IV. push,
      OR
   2. Amytal 3¾ gr. I.V. push,
      OR

TABLE III

ANAPHYLAXIS
FLOW SHEET

TREATMENT

Date — Time	
Recumbent Position	
Elevated Legs	
Hyperextended Neck & Head	AIRWAY
Airway Tube w/oxygen 4 (6) liters/min.	
Suction	
Ventilatory Assistor Apparatus	
Keep Mouth Open	
?? Endotracheal Intubation	
?? Tracheotomy	
0.5 cc into base of tongue	ADRENALIN
Subsequent injections I.V. or subcutaneous	
Injection into puncture site or insect sting	
Dilute 1cc/1:1000 in 10 cc/physio. saline I.V./5 min.	

15-30 mg. Wyamine I.V. Push	SHOCK
Levophed + Regitine in 1 liter D5W I.V.	
Foley catheter 40 cc/hr of urine	
Seconal — or	SEDATION
Amytal — or	
Valium	
10-20 cc STAT I.V. Push (slow)	AMINO-PHYLLIN
10-20 cc/liter D5W q 12 h I.V. drip	
Hydrocortisone 300 mg. I.V. STAT — then	STEROIDS
100-200 mg. q 6 h I.M. or I.V. drip	

   3. Valium 5-10 mg. I.V. or I.M.

E. *Antihistamines*
   1. Benadryl 50-100 mg. I.V. q 6 h.

F. *Steroids*
   1. Hydrocortisone 300 mg. I.V. STAT.
   2. Then, 100-200 mg. q 6 h I.M. *OR* by I.V. drip.

G. *Aminophyllin*
   1. 10-20 cc STAT I.V. push slowly (250-500 mg.) and then,
   2. 10-20 cc per one liter D5W q 12 h I.V. drip.

H. Loosen constricting clothing.

I. If glottic closure, insert two 12 gauge needles into trachea via thyrohyoid membrane.

J. Early tracheostomy.

K. Give lastly Chlor-trimetron 10 mg. I.M. at the injection site.

L. IPPB with Isuprel. (0.5 ml. Isuprel Isoproterenal and 1.5 ml. saline.)

M. For cardiac arrhythmias, Procainamide 250-500 mg. every 4-6 hours.

N. Heparin ampules of 100 mg. times 3 given I.M. *OR* I.V. over two-minutes period to control edema of tracheo-bronchial tree.

O. Neutrapen (800,000 units) I.M. — if penicillin is cause. It *acts slowly*.

P. If the result of stinging insect, 0.10 cc of No. 9 dilution (1:40,000,000) of the mixed sting insect antigen.

Q. If insect sting, remove stinger by flicking (not squeezing).

The treatment of mild and local reactions is summarized in Table IV, and the treatment of a related problem — giant hives or urticaria with severe itching — is summarized in Table V. For prevention of anaphylaxis, all patients should be questioned regarding previous drug reactions with specific reference to whatever material is to be administered. It should be remembered that direct skin testing — as an attempt to corroborate the history — may itself produce a fatal reaction. Therefore, when the history is questionable and skin testing is indicated, facilities for treatment of anaphylactic reactions must be readily accessible. These facilities include agents for parenteral administration (epinephrine hydrochloride, aminophylline, diphenhydramine, levarterenol, hydrocortisone hemisuccinate, isotonic saline solution), as well as instrumentation for endotracheal intubation or tracheostomy. Also required is the establishment of an intravenous infusion in the extremity opposite the one to be tested, the infusion to be maintained for 30 minutes after testing. □

TABLE IV

TREATMENT OF LOCAL OR MILD REACTIONS

1. Apply a tourniquet proximal to the site of reaction on an extremity. Relax occlusion for one minute out of every three.
2. Inject 0.012 ml of 1:1000 aqueous epinephine hydrochloride subcutaneously into the site of reaction.
3. Inject 0.3 ml of 1:1000 aqueous epinephrine hydrochloride subcutaneously into the opposite extremity.
4. Administer 50 mg of Benadryl orally or intramuscularly, depending on the severity of the reaction.
5. Administer 1 tablet of Theophylline-Ephedrine-Phenobarbital (Tedral).
6. Observe patient until the reaction begins to subside (usually 30 to 60 minutes).
7. If there is no response within 30 minutes of observations, additional measures must be taken. If the reaction is widespread, give Decadron 4 - 9 mg intramuscularly.

TABLE V

TREATMENT OF GIANT HIVES OR URTICARIA WITH SEVERE ITCHING

1. Benadryl 50 - 100 mg I.M., followed by oral antihistamines.
2. Observation by a physician to determine whether oral steroids or injectable steroids are indicated.
3. Topical steroid preparations are also administered.
4. Epinephrine (Adrenalin) 1 cc of 1:1000 aqueous I.M.
5. If necessary, 8 mg of Decadron IV with 150 cc of D5W.

KEEPING UP WITH AND MANAGING PATIENT EXPECTATIONS: IMPLICATIONS FOR IMPROVED RISK AND PATIENT MANAGEMENT

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All health professionals, by now, are well aware of the many pressures for "keeping current." Such pressures have been created by the growing torrents of important new data, information, "breakthroughs" and other often complex developments in the health and health-related professions. Most health professionals are responding accordingly by investing considerable time and energy to at least cope with this growing flood of information. Contemporary health professionals have been forced to sift out the often diffuse elements of these new developments in their own and related disciplines which seem to have some significant relevance to the conduct of their practices. Similarly, most health professionals are being ever more frequently confronted with increasingly assertive patient expectations. In fact, in many extreme cases, these patient expectations have become patient demands which, if not deftly addressed, have been and can be a major motivating factor in the patient's institution of malpractice litigation. However, few health professionals, except those who have learned "the hard way," adequately comprehend the subtle and perhaps critical interrelationships between "keeping up" in the conventional sense, i.e., with new clinical, scientific, technological, and practice management developments, and "keeping up" with individual patient and patient group expectations.

Furthermore, few health professionals have applied an understanding of these interrelationships by managing patient expectation as a key aspect of improving not only patient-physician relationships, patient compliance, and resultant treatment outcomes but also practice risk management. Therefore, it is the principal objective of this paper to articulate and develop these interrelationships between the physician's "keeping up" and patient "expectations" and to elaborate on their implications for risk management. In fact, a case will be made for your actively monitoring patient expectations and managing them as an increasingly important "new development" in patient management and as a major commitment activity of overall practice management.

I. "KEEPING UP WITH" PATIENT EXPECTATIONS: THE RATIONALE

An often neglected but critical aspect of "keeping up" is the requirement that the health provider actively inventory and assess the changing expectations of each individual patient and family in the context of each patient management episode. This requirement resides in the larger context of the health provider's needing to understand and respond to the nature of changing expectations of his health practice patient population in the aggregate and, indeed, the health care expectations of the greater society. These various evolving, levels of patient expectations, in fact, encompass what the various patient individuals and groups have been increasingly regarding as their health care "needs" including how these needs ought to be properly addressed by health care providers. Indeed, to the alarm of many health providers and other concerned observers, many of these individual or group health "needs" are being promoted through many channels as a health care "rights" (even before many of these have been clearly established as "needs"). Hence, health providers are being challenged to innovately and resourcefully respond to these emerging health care expectations become "needs" and "rights."

In a broader sense, a convincing case could be made that, what has been happening here has been, in lieu of adequate principles, standards, and requirements of health practice having been established and abided by mutual health provider and consumer agreement, health consumers have been developing their own expectational standards of practice, identified in the extreme as "needs," "rights," and even "guaranteed" treatment outcomes. Any health provider who can at least begin to subscribe to this latter contention as a real phenomenon or movement, however unsettled, frightened and threatened by it he may find himself, should begin "keeping up with" and responding appropriately to this complex spectrum of patient expectations.

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This "keeping up with" and responsiveness to patient expectations has become ever more essential in the face of, like it or not, the fact that even primary health care provider-managers, no longer exclusively "own," shape, or control their patient's expectations and attitudes regarding health status, health care and its management. Even so, many physicians and other health care providers are relatively unconcerned about or are simply passively resigned to this. Many go on blithely with the turn of the century mentality wherein the old family doctor friend orchestrated everything of health care concern in patients' minds. However, it is indeed a "new ball game" with a growing deluge of fiction and non-fiction books (e.g., home health guides, health provider biographies and autobiographies, etc.); magazine and newspaper articles and columns (e.g., *Reader's Digest* "I Am Joe's . . . ."); daily syndicated columns by Drs. Salk, Spock, Smith, Stare, Thosteson, Reuben; many daily newspaper feature stories on health and health issues, etc., etc.); TV programs (e.g., Ben Casey, Marcus Welby, Emergency, many doctor and hospital-oriented "soap operas" and other series); educational TV programs on health and health issues; etc. All of these heavily influential, daily forces in aggregate probably virtually totally eclipse the impact of the personal physician in shaping patient and societal attitudes and expectations regarding health.

Whether or not they may be scientifically accurate in their presentations of health information and whether or not they have been developed by qualified, well informed health professionals, these media in their collective impacts, along with those of many other forces, including the advice and counsel of family members, neighbors, peer health group members, and attorneys must be appreciated as a most potent aggregate of forces competing with those of health providers in molding health consumer attitudes and expectations. The competition in this domain, once dominated by the health provider, has become fierce and treacherous for health provider and consumer alike. There is much more opportunity for confused, unfounded, unrealistic, and otherwise dangerously inappropriate patient expectations to develop. The adverse impact of these diverse expectations-shaping forces run amuck may be dramatically exemplified in the recent waves of diet or weight control fads, often doctor-initiated, which have swept across the U. S. and have culminated in the amphetamine abuse, the intestinal bypass, and, most recently, the liquid protein diet scandals and disasters.

Remember too, that these patient expectations and attitudes-shaping forces exercised by non-health professionals can have significant impacts not only in anticipation of impending health care management intervention but also after health care services have been rendered. Much of the recent increase in malpractice activity may be traceable to situations where the patient, with the real or imagined undesirable treatment outcome, could readily justify, at least in his own mind, seeking malpractice redress, based on expectations shaped predominantly or almost exclusively by such non-health professional forces. Here the health professionals have defaulted to the competition in not bringing aggressively to bear their opportunities to more appropriately shape patient attitudes and expectations and thereby leaving themselves subject to a double jeopardy, that of the possibility of poor treatment outcome followed by malpractice suit.

It must also be kept in mind that many patients now have reasonably ready access to many heretofore relatively unavailable other health professional counsel, opinions and reference materials like textbooks and outlines of medicine, PDR's, Merck Manuals, home guides and handbooks on many aspects of health care and the like. The patient may have even deliberately "boned up" intensively beforehand to "second guess" and/or test your medical or health care acumen and management competence. Additionally, either a relatively well informed or a basically misinformed patient, who has or thinks he has done some homework, and thinks he knows as much as or more than the physician may be coming in with a "chip on his shoulder." He may be, in effect, waiting to legally pounce on or entrap the physician who ignores, discounts or otherwise depreciates his "knowledge" and expectations. Such health provider inattentiveness to patient expectations sets the malpractice "snare" or "trap." Such a "snare" is effectively triggered when the patient sees himself as having incurred an undesired result or outcome from his health care management program. Of course, this "trap" or "snare" can in many cases be avoided or disarmed early on by the wary and astute physician who is alert to and actively elicits the signs and symptoms of the high risk litigious patient. What these signs and symptoms may be and the ways in which such snares or traps can be disarmed or avoided, from refusal to treat to utilization of counselling and placebos, have been very adequately treated elsewhere in the literature.

II. IMPLICATIONS FOR IMPROVED RISK MANAGEMENT:

Why then is "keeping up" with patient's health care expectations so critical an aspect of medical malpractice prophylaxis or risk management? It is contended: that if the primary health provider consistently commits himself to determining, as fully as possible, what in fact is the milieu of changing patient expectations during each patient management episode and deftly shapes and manages these, then there is little or no basis or potential for conflict much less malpractice confrontation. Of course, as an integral major part of the patient-physician relationship, the physician not only should assess his own reasonable expectations, including formal prognosis, regarding patient treatment outcome, but also should mutually shape and share all case expectations with the patient and document these processes as well. This advisory should pertain whatever the case outcome, real or imagined, desirable or undesirable, however "unexpected." If, however, the primary health care provider does not insist on and commence working toward achieving the advised common or shared reasonable expectations with the patient, even before the laying on of hands, and should there result an undesirable outcome, real or imagined; then there is ample potential for serious consequences. Such consequences include loss of a patient and his family as patients, as well medico-legal conflict which, in the extreme, occurs as malpractice litigation or the pressing of assault and battery charges!

In impatience and frustration, one might ask how is it humanly possible to get a reasonably accurate reading on the "baseline expectations" of upwards of 2000 patients in any given full time practice much less keep them all current? Also, who can become and remain that patient expectations "current" even exclusive of the always pressing time limitations and other constraints? It is humbly suggested that a reasonably adequate and appropriate baseline and subsequent readings of each patient's expectations regarding his illness and/or disease, his treatment program, and his treatment outcome(s) could be elicited from each patient. Such patient expectations should be elicited after the initial workup as well as after each follow-up history and physical (or laboratory) evaluation and *before* the new or revised diagnosis *and* the new or revised projected plan of health care management is shared with the patient. This can be accomplished by asking the unprompted patient, "*what type of illness or disease he then thinks he or she has and what this means for him/her as well as what he thinks can and should be done about it by the doctor and his health team?*" Note this goes beyond the eliciting of the "present illness." It is felt that in most cases the major salient features of any given patient's expectations can be elicited in a very few minutes. This can and should be done in a non-leading questioning and tactful manner which also reflects the physician's interest in and concern for the patient, the patient's appreciation of his own health status, and his projected course of health care.

In addition, this aspect of the patient interview should be accurately documented, cosigned by physician and patient, witnessed, and dated on standard patient records media or recorded on tape as a formal complement to the instrument(s) of informed consent as these might be indicated. Such an exercise represents not only sound legitimate primary "defensive medicine" but also, perhaps even more importantly, sound patient management. It therefore should prove well worth the not inordinate investment of time and effort. Consider this in the lights of the "tradeoffs" of increasing astronomical malpractice premiums, the unavailability of malpractice coverage, and increasing malpractice "troubles" versus seeing a few less patients each day. Obviously, diminishing returns have long since set in for many practitioners for them to lightly rationalize away or dispense with such well-advised recommendations for risk management.

Later, even if malpractice proceedings should eventuate, such a formal documented record of the patient's own statements expressing his expectations can be brought to bear as powerfully convincing evidentiary material. It is available to the physician as documented evidence of his active concern for and interest in the patient and his ongoing attempts (beyond the patient just automatically signing off on an informed consent or release form) to bring, through ongoing patient (re)education, the patient's expectations in line with those of his physician. The obvious goal here has been the achievement of a continuously improved basis for working together (i.e., informed compliance and cooperation) toward realizing a mutually desired and "expected" optimal treatment result. Such documentation of "expectations" would serve as direct and convincing corroborative evidence of the physician's attempt(s) at utilizing, appropriately and in mutually consistent ways, the instruments of informed consent, release, exculpatory agreement and the like. All this as a concerted body of documented evidence of sound patient management activity would further establish and reinforce the health provider's soundness of intent, responsiveness, conscientiousness, and judgment. This would pertain even in the face of the patient's refusal or reluctance to adopt realistic expectations, congruent or at least proximately compatible with those of the health provider.

With the following as key facets of the documentedly complete patient record: (1) written description of the patient's baseline expectations documented in the original workup with appropriate updates, (2) written or taped documentation of the patient's evolving expectations, (3) written or taped record of the physician's ongoing attempts and results at bringing patient and physician expectations into alignment as integral parts of the "progress notes," and (4) the patient and physician signed, dated, and witnessed instruments of informed consent and other essential formal "agreements," where appropriate; the physician ought to find himself much better prepared to mobilize an exceptionally strong defense of his professional judgment and case management at any level of the preliminary or advanced adjudicatory processes.

Since persistently at variance patient-physician expectations, whether actual or contrived, coupled with undesirable outcome, have been a major impetus for medico-legal litigation, it is particularly appropriate that such explicit documentation be developed and kept readily available. Such evidence should prove to highlight these critical persistent differences in understandings and expectations among all the major parties involved. Well documented "patient expectations management" should inventory in some detail how the physician has striven to properly resolve these differences through patient education and counselling. The adoption and implementation of this sort of risk management regimen in each case should help considerably to preclude the development of inappropriate expectations and misunderstandings and attendant disorientation and confusion during the patient management cycle itself often leading to patient-physician disagreement and confrontation. The record should clearly reflect which critical understandings and expectations have been agreed upon, which have been only marginally agreed upon, and which have been at significant variance. It should also reflect when this in or out of agreement status has significantly changed with reference to certain major areas of expectations, if and when the involved respective expectations change.

CONCLUSIONS:

The primary health care manager cannot expect to totally offset or counterbalance the many patient expectations-shaping forces cited above which are external to the physician-patient relationship. However, he must; in the best interests of optimal patient management and improved risk management; attempt to inventory, assess and effectively respond to the major relevant features of this complex of externally imposed patient expectations. Well-advised physician response to these major expectations should probably include simply reconfirming and meeting them if they are reasonable, realistic, and otherwise appropriate as well as compatible with those of the physician and prevailing standards of practice. The physician should also frankly but tactfully share his expectations with the patient as well as formally document the evolving major expectations of all involved principal parties. He should also record efforts toward this mutual sharing of expectations as well as what observable impacts such efforts have had. However, if there are significant time variable discrepancies between one or more of the major expectations areas held by the patient and by the physician, the physician should attempt to resolve these discrepancies through aggressive patient health education and counselling. This process should be undertaken even before active therapeutic or even dangerous diagnostic intervention is implemented if such intervention can be safely and otherwise justifiably deferred in order to resolve these expectational differences first.

Health providers are well advised to document the evolving differences in expectation, if, when, and as they arise among all the principals involved. Health providers should also document their deliberate efforts to resolve such critical differences. The attempted resolution of these differences as managed and implemented by the involved health providers should rigorously conform to the highest standards of sharing relevant, accurate, valid, and useful information toward resultant improved understanding through health education and counselling of patients and their families.

Inadequate, erroneous or otherwise marginal or inappropriate information and understanding when conveyed to or allowed to persist and evolve in the minds of patients can mean disaster for patient and physician alike. Patient trickery, subterfuge, and coercion to effect anything in the patient management setting, much less agreement among patient and physician expectations, is probably extremely ill-advised for it can "backfire" catastrophically.

The above recommendations and their rationale are shared with the health provider as additional useful insights and related approaches toward ever improving risk management, in particular, and overall patient management, in general. Perhaps, if such recommendations were to come to be conscientiously and widely honored, the health professions could then reasonably hope not only to recoup their former positions as far and away the greatest force shaping patient and societal health expectations but also to experience an abatement of a major class of troublesome and costly litigation. □

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1979 ANNUAL MEETING OF THE AMERICAN MEDICAL ASSOCIATION

C. TUCKER WESTON, M.D.*

The 1979 Annual Meeting of the American Medical Association was held at the Chicago Marriott Hotel, July 22-26, 1979, and was attended by a full delegation from South Carolina, with enough representatives to assign one man to each Reference Committee for monitoring all sessions. In attendance from the SCMA were Senior Delegates C. Tucker Weston, M.D., and Waitus O. Tanner, M.D.; Alternate Delegate Donald G. Kilgore, Jr., M.D., and Substitute Alternate Delegate John C. Hawk, Jr., M.D.; Chairman of Council Euta M. Colvin, M.D.; President Harrison L. Peeples, M.D.; President-Elect Halsted M. Stone, M.D.; Secretary William H. Hunter, M.D.; and SCMA Executive Director Charles Johnson.

The Convention was of special interest because of our Delegation's activities in promoting John Hawk for election to the AMA Council on Constitution and Bylaws, with the hospitality room entertainment and solicitation of support from the entire House of Delegates which culminated in John's successful election. Our special thanks and appreciation to the wives in attendance who assisted with the SCMA hospitality room on Sunday, Monday and Tuesday nights.

We were all very pleased that Waitus Tanner had the honor of serving as a member of a major reference committee at this meeting.

It was the opinion of all South Carolina participants that this was a very interesting and successful convention. The trend towards conservatism in the House of Delegates is continuing. There has been a rapid change in the membership with the addition of the 56 specialty society members. We are convinced that we should fully participate and support the AMA as the umbrella for organized medicine. Only by the individual physician's support at the grass roots can the real voice of the American physician be heard at the national level.

Our congratulations to Robert B. Hunter, M.D., who was elected President-Elect at this meeting. You will recall Bob's visit to the 1979 Annual Meeting of the SCMA.

Your entire Delegation expresses appreciation to you, the membership, for the privilege and honor of representing the SCMA at the American Medical Association. We are presently making plans to represent the SCMA in Honolulu at the Interim Meeting in December, 1979. I have been asked to Chair Reference Committee G, and look forward to serving in this capacity.

The new format will be used for this report, with individual representatives detailing the activities which took place in the Reference Committees to which they were assigned. These individual reports follow.

REFERENCE COMMITTEE ON CONSTITUTION AND BYLAWS — John C. Hawk, Jr., M.D.

Principles of Medical Ethics:

It was a foregone conclusion that the proposed new "Principles of Medical Ethics" brought in by the Ad Hoc Committee would be the subject of considerable debate. The committee report made five recommendations; recommendation number three was clearly desirable, essentially non-controversial, and was passed without dissent. This recommendation called for a change in the Bylaws which would make it perfectly clear that the House of Delegates and *not* the Judicial Council has the prerogative and duty to establish the Principles of Ethics for the AMA.

The Reference Committee noted that state associations, component societies and specialty societies had not had an opportunity to study in detail and to comment upon the Report of the Ad Hoc Committee and, therefore, recommended "that the remainder of the Final Report of the Ad Hoc Committee on the Principles of Medical Ethics be distributed to state medical associations, component medical societies and specialty societies for study and consideration by their respective Houses of Delegates; and for possible revision by the Ad Hoc Committee; and for presentation of a progress report to this House of Delegates at the 1979 Interim Meeting and a final report at the 1980 Annual Meeting".

In the Reference Committee hearings, there was much comment and some inquiry about a so-called "red-lined" version of the old Principles of Ethics, said to have been developed by legal counsel as the minimum changes in the Principles of Ethics which would avoid further difficulties with The Federal Trade Commission and other government agencies.

The Reference Committee, presumably with this in mind and in response to Resolution 71, recommended adoption of a substitute Resolution which states: "RESOLVED, that a proposed amended version of the American Medical Association Principles of Medical Ethics, containing only such minimal changes as may be necessary, be submitted by the Board of Trustees to the House of Delegates at this meeting for consideration at the 1979 Interim Meeting".

Surprisingly, this was defeated 132 to 123, but in any case, the "red-lined" version was made available for consideration by state associations, specialty societies, etc. between now and the Interim Meeting.

Chiropractic:

Tied to the issue of the Principles of Ethics was the question of the AMA position on chiropractic. Three pending anti-trust suits against the AMA were undoubtedly high in the consciousness of the delegates when they considered the Board of Trustees' recommendations, contained in Report UU. The softened stand against chiropractic was not to the liking of many delegates, but was adopted by the House for what was referred to as "prudent" legal reasons. It was noted that chiropractors are licensed in all 50 states. The Board report states: "It is better to call attention to the limitations of chiropractic in the treatment of particular ailments than to label chiropractic an 'unscientific cult'."

Other Matters:

In other actions on matters considered by this Reference Committee, the House of Delegates:

1. Accepted recommendations of the Board of Trustees to grant representation in the AMA House of Delegates to three new specialty societies and to deny representation to one. This brings the total number of specialty societies represented to 56. The House also adopted a Rocky Mountain Conference amendment asking the Board of Trustees to place a moratorium on new specialty society representation in the House of Delegates until such time as Section 8.40 of the Bylaws can be studied.
2. Adopted the Texas delegation recommendation to accord voting privileges to medical students on Councils and

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Committees at the discretion of the individual AMA Council or Committee on which they serve. At the same time, the House voted not to adopt the Student Business Section Resolution asking that medical students be accorded voting privileges on AMA Councils and Committees on which they serve, regardless of whether the committee so desired.

REFERENCE COMMITTEE A — Waitus O. Tanner, M.D.

Medicare:

A substitute Resolution for Resolution 26 was adopted regarding Medicare reimbursement for laboratory fees. The Substitute Resolution stated that the AMA should communicate with HEW to request reimbursement for lab tests performed in physicians' offices on an equitable basis rather than the least paid to automated laboratories, and reimbursement of specimen handling fees would be taken care of also. This was referred to the Board of Trustees for implementation.

Resolution 34, dealt with misrepresentation to the public of Medicare reimbursement rates. Adopted was a resolve that the HEW Medicare program misrepresentation to the recipients regarding reimbursement levels for physicians' fees should be investigated. Resolution 39 concerned Medicare payments for CT scanning. Adopted was a resolve that the AMA officially oppose Medicare payments directives regarding CT scanning, protest HEW abridgement of UCR fee principles and PL 8997, and that HEW be advised that the reimbursement policies can result in limiting worthwhile technological advances.

Resolution 19 commended the AMA for its support of legislation authorizing physicians to receive copies of communications sent by Medicare fiscal intermediaries to physicians and patients.

Rural Health Clinics:

Report J of the Council on Medical Services, Resolution 124, dealt with rural health clinics and their reimbursements. Adopted was a resolve that the Council on Medical Services be instructed to continue monitoring quality assurance and reimbursement procedures in federally funded health clinics, and to report to the House of Delegates in 1980. Resolution 142 reaffirmed AMA's support of the concept of UCR, with HEW to be informed of this action.

HSAS:

Several resolutions were studied regarding Health Systems Agencies. Some wanted to eliminate them altogether by various means, including cutting off of federal funds. Adopted was a resolve that the AMA petition the U. S. Congress to eliminate or drastically reduce federal funding of health planning and that the AMA encourage each constituent state medical association and component county medical societies to actively urge physicians to express their views in a positive manner to each member of Congress. Other Resolutions considered the problems and the makeup of the HSA Boards and indirect providers, with the contention that this category of indirect providers should be eliminated.

Other Matters:

Concern was expressed in Report G of the Board of Trustees on the conflicts in the National Health Service Corps program, especially in the re-defining of health shortage areas. The Board will seek legislative amendments reinstating local, professional medical societies as participants in the National Health Service Corps program.

Resolution 2 from South Carolina concerning the collection and dissemination of data regarding the cost of medical care was not adopted because it would be too expensive and what is proposed is being done in a different manner by the AMA staff already.

Resolution 6 was adopted which resolved that the AMA endorse universal health insurance claim forms for submitting Workmen's Compensation claims.

REFERENCE COMMITTEE B — C. Tucker Weston, M.D.

Cost Containment:

The AMA House of Delegates supported Resolution 7 which recognized the effectiveness of the voluntary health care cost containment effort, and opposed HR 2626, the Hospital Cost Containment Act. The House referred to the Board of Trustees for further study a Resolution advocating a separate National Health Department.

Discrimination Against Teaching Hospitals:

The House urged the introduction of legislation to repeal Section 227 of PL 92603 in order to eliminate or reduce the inequity of differential payments for services rendered by physicians in teaching hospitals.

National Health Insurance:

With regard to the issue of National Health Insurance, Resolution 62 of the AMA 1978 Interim meeting was reaffirmed. Reports N and XX of the Board of Trustees were adopted, including the need for the AMA to continue to play an active role in further Congressional consideration of National Health Insurance. Substitute Resolution 79 was adopted that the AMA continue to state in its publications, testimonies and to its spokesmen the extent of its public and private health care coverage of the American people.

Federal Trade Commission:

The House approved a Resolution that the Board of Trustees monitor activities of the FTC and initiate and support remedial legislation where advisable. With regard to the FTC staff recommendation that Blue Shield and other similar medical plans have the physicians removed from their Boards, the Resolve was that the AMA continue to oppose proposals that curtail communications or participation on the part of the medical profession with the governing boards of health care plans.

It was brought out that all physicians should be alerted to actively participate in the forthcoming FTC hearings because if they do not participate in the initial ones, they will be barred from later hearings.

Other Matters:

The Board of Trustees was asked to continue monitoring gasoline regulations to allow enough gasoline for physicians. The House voted continued opposition to equal payment under Part B of the Medicare program to ophthalmologists and optometrists.

A Resolution was adopted to encourage state societies to solicit views of medical students when legislation affecting students is being considered.

The House voted opposition to the establishment of a Doctors Draft. Private medical insurance for uninsurables was supported, with the AMA to work with the insurance industry to develop mechanisms to make such coverage available.

Report U of the Board of Trustees was adopted and urged continuing the monitoring of and efforts to support the physicians legislative contact programs throughout organized medicine. Support was voiced for proposed legislation to increase the Keogh Plan benefits to realistic levels.

REFERENCE COMMITTEE C (MEDICAL EDUCATION) — Harrison L. Peeples, M.D.

Of General Interest:

With regard to Continuing Medical Education (CME), the House adopted resolutions and Reports that (a) give us a new definition of continuing medical education; (b) deplore excessive charges for CME courses which exploit physicians and urge state societies and the AMA to consider cost in the accreditation process; also to acquaint physicians with programs that will help them meet their particular needs at a reasonable cost; (c) urge the AMA Board of Trustees to invest funds in effective, self-instructional education programs; (d) continue to urge physicians to *voluntarily* participate in CME; (e) the AMA withdraw from the Liaison Committee on Continuing Medical Education and resume the AMA's previous responsibility for the voluntary accrediting of continuing medical education through the establishment of a Committee on Accreditation of CME of the Council on Medical Education. The state medical societies will be immediately recognized as the accrediting bodies for institutions and organizations providing local and inter-state programs for CME.

Of Interest to Medical Schools:

The following resolutions and reports were adopted: (a) the House of Delegates of the AMA supports the principle that the best qualified student be admitted to medical schools . . . and reaffirms that it is desirable for the student body to reflect a wide spectrum of social and economic backgrounds as stated in "Function and Structures of Medical Schools"; (b) the AMA adopt and help implement the principle that all medical schools and others include in their curriculum subject matter which would help educate students in school and graduates in training, practitioners and graduates in CME programs in *medical economics* and *cost containment* and would bring about an understanding that the function of organized medicine *is safeguarding the interest of the patient and discharging the obligation of the medical profession.*

Of Interest to Medical Students:

Resolutions and reports adopted by the House would have (a) the AMA support federal legislation and urge development of model state legislation which would provide increased financial assistance to medical students; (b) the AMA support the concept of deferral of repayment of principal for medical education loans until after graduate medical training; (c) the series of due process guidelines for medical students be used as stated in Report B of the Council on Medical Education (this report available from the AMA on request).

There was strong support for funding of preventive and community medicine residency training programs and support to expand graduate degree programs.

The armed services and national guard have been having increased difficulty in continuing their training program and in recruitment of adequate personnel. The House of Delegates reaffirmed its support of actions designed to support the services as stated in substitute Resolution 1 and substitute Resolution 3. These would encourage recruitment and funding for training programs at a rate sufficient to meet the need.

REFERENCE COMMITTEE D — Halsted M. Stone, M.D.

Jurisdictional Disputes:

The first group of Resolutions considered was in the area of jurisdictional disputes between specialists. The Resolutions supported the concept that a physician trained to a procedure be allowed to do this procedure if his competence was satisfactory and his audits bear out this competence. This was supported.

Hospital Cost Control:

The idea was presented that the hospital board is in concert with the medical staff to approve or disapprove hospital programs, services and expenditures. Another Resolution asked that the JCAH study the financial impact of any new regulations and the impact upon volunteer staff time that any new regulations would have. It was requested that all new regulations show how they would improve patient care. Joint surveys were requested to help reduce cost. Report C on hospital cost and hospital charges in relationship to these costs was referred for further study.

Medical Liability Insurance:

Report KK dealt with medical liability insurance as a mandatory requirement for staff appointment. It was concluded that each staff should decide this for themselves, but there was great concern expressed that staffs should not go without insurance and leave the hospital with the total financial burden. It was noted that the hospital boards may require staff to have liability insurance. This was referred to the Board of Trustees for further study.

Hospital Accreditation:

It was asked that hospital accreditation be increased to three years from two years in another Resolution. This was to be considered from the cost containment point of view in that the cost of accreditation is quite considerable. This was adopted by the House, but it was not felt that the JCAH would go along with it.

Nursing Homes:

Nursing homes were discussed at great length. It was asked in one Resolution that direct admissions to skilled nursing homes be allowed, thus bypassing the three-day hospital stay which is now required. This would require a change in the federal regulations. It was felt the concept was good and everyone was in agreement, but it was not thought that it had much change of succeeding with the federal government. The Resolution was adopted with amendment.

Autonomy of the Hospital Staff:

Autonomy of the hospital staff was requested in dealing with hospital boards and hospital administrators. It was generally felt by those discussing these ideas and Resolutions that this almost represented a union approach and was not felt to be a good idea.

JCAH Requirements:

The JCAH was asked to strengthen the wording in its requirements dealing with hospital boards to assure representation by a physician staff member with a vote. A Resolution from Georgia requested the JCAH that only physicians be allowed to write on the patient's chart in the section labeled "Doctor's Progress Notes."

Hospital Emergency Room Services:

Categorization of hospital emergency room services came in for considerable debate. This concerns level one, two, three and four. In level one, the 24-hour in-house anesthesiologist and the 30-minute response time by medical staff was discussed at great length. It was pointed out that in the 1980 JCAH regulations, two-way voice communication will be suitable for the initial consultation. It was further noted that any hospital may choose whatever category it desires, and none will be required to take any category unless they so desire.

There was a lengthy report on hospital emergency services in which the idea was presented that it was good to attempt to do something but that nothing much could probably be accomplished in this particular area. It was with a great deal of pride that I was able to point out what South Carolina has accomplished to date with our proposed triage system. Everyone was most interested.

REFERENCE COMMITTEE E (SCIENTIFIC AFFAIRS) — Donald G. Kilgore, Jr., M.D.

Tobacco and Nuclear Energy:

Report C of the Council of Scientific Affairs concerning smoking and health was adopted by the House, along with two other Resolutions opposing television advertising of tobacco and the commendation of those publications which have refused to accept cigarette advertising.

A group of recommendations were considered concerning the study, use and disposal of radioactive waste. The major thrust of Resolution 171 adopted and called for a study of the inherent safety of nuclear power and a report back to the House at the 1979 Interim Meeting.

The discussions on the use of tobacco and nuclear energy consumed most of the hearing time. Many of the witnesses properly confined their discussion to provable scientific facts and quiet, reasoned argument. However, I regret to report that many witnesses apparently forgot they were physicians. The use of abusive language toward those who differed from their view, the use of testimonials from people unqualified to judge scientific evidence (reminiscent of advertising of chiropractic and medicine show charlatans), and a fanaticism which would deprive some people of their rights under the Constitution, was rather disappointing to observe. A few hours later, I was relieved to see one of these antismoking zealots intoxicated to the point where he could hardly stand. The reason I was relieved was that I was glad to see that such a perfect individual had at least one fault.

Pharmacology:

The House adopted the recommendation that the AMA inform medical students that the AMA will provide them with a complimentary copy of "AMA Drug Evaluations" upon request, during the year they have their formal course in pharmacology. The House also approved the recommendation that the AMA recommend the use of Syrup of Ipecac in the event of accidental poisoning, but that the drug not be administered before consulting a physician. A request that the AMA establish policy dealing with the prescribing of drugs for amateur athletes was referred to the Board of Trustees for further study and report to the House.

Other Matters:

Report A of the Council of Scientific Affairs on indications for aorto-coronary bypass graft surgery was approved by the House, with a caveat stating that reports such as this do not establish standards of medical practice or care because these standards change and evolve day by day in locality by locality.

A Resolution asking the AMA to adopt a statement of the Uniform Brain Death Act of the National Conference of Commissioners on Uniform State Laws was referred to the Board of Trustees. Report F, a commentary on the final report of the President's Commission on Mental Health received mixed reactions, and was referred to the Board of Trustees. Resolution 150 was adopted, in which the AMA reaffirmed its policy favoring the multiple approach to collection of blood, thereby encouraging blood banks to employ incentive programs proven successful in recruiting voluntary donors, and urging the American Blood Commission to revise its 1977 action advocating a single approach to blood collection. It was further resolved that should the American Blood Commission continue to advocate the singular approach, the AMA would re-evaluate its membership in this body.

Report E was adopted, urging that the AMA implement a six-point program designed to emphasize the seriousness of alcoholism and the importance of the physician's role in the prevention and treatment of this disease. Another report was adopted which affirmed the desirability of physician education programs at all pre-collegiate level schools. At the same time, the House approved a recommendation calling for AMA opposition to that portion of the Rehabilitation Act of 1973 (PL 93-112) which provides that students who have lost an organ, limb, or appendage but who are otherwise qualified may not be excluded by their respective institutions from participation in contact sports.

REFERENCE COMMITTEE F — BOARD OF TRUSTEES — William H. Hunter, M.D.

Membership and Finances:

Membership in 1978 in the AMA has increased by about 1,000 from 1977. Of interest is the following years: 1975 — 162,400 members; 1976 — 151,700 members; 1977 — 146,600 members; 1978 — 147,700 members. Between 1975 and 1976, there was a dues increase which caused a corresponding decrease in membership for those years. Recently, there has been a rather marked increase of students and residents. There are 60,000 members of state and local societies who are not AMA members. Forty percent of the M.D.s in the United States now belong to the AMA. In 1973, 58 percent of the M.D.s in the United States were AMA members. Inflation is eroding the budget rather badly. The 1979 budget will be $54,382,000 which is up from 1978's budget of $52,000,000, the increase due totally to inflation.

The AMA plans to build a one million dollar office building in Washington, which will pay for itself and in the long run be a good investment. The curves of revenues and expenses will cross during 1981, and at that time, to keep the AMA out of the red we must do one of three things: obtain new members, increase the dues, or we must cut costs.

Reserves of the AMA are adequate and are projected to $53,258,000 in 1980. The AMA has 67 million dollars in total assets. Investments in the AMA are returning over nine percent. The AMA owns 10 percent of the Chicago Marriott Hotel, having invested 2.2 million dollars therein. At this time, it could be sold for eight times the investment.

Report of the Chairman of the Long Range Planning Committee, Dr. Broadaway:

Dr. Broadaway emphasized strongly that it is the feeling of the Long-Range Planning Committee and the Board of Trustees that at this time there is a crisis in the membership in the AMA. To meet this problem, there were three proposals: (1) that the AMA should be an umbrella for all medicine, (2) the AMA is to bill and recruit directly, and (3) that all state society members should become members of the AMA. The Board of Trustees reported on these proposals with the following brief note: "The Board also endorses the Council's recommendation for renewed efforts to strengthen AMA's umbrella role. The Council's analysis clearly confirms what has been a conviction of the Board of Trustees and the House of Delegates for many years, that the AMA is the only organization that can perform a truly umbrella role within medicine. The Council's analysis also presents telling arguments for incorporating a forum of the 'Organization of Organizations' concept into the AMA's organizational configuration. The Board urges the House of Delegates to endorse the concept and to ask the Council to continue its development work on this proposal." The House of Delegates asked the Board to continue its investigation into the matter and ask for comments from state associations.

REFERENCE COMMITTEE G — Euta M. Colvin, M.D.

Professional Standards Review Organizations:

Testimony was generally in support of the PSRO program. However, considerable concern was expressed over the program's increasing emphasis on cost containment rather than quality assurance. The Reference Committee felt that such corruption of the intent of the PSRO program is a real and apparent danger, and notes that the AMA may wish to reconsider its support of PSRO if cost containment becomes a predominant program objective. It was felt that the Association should continue to support the PSRO program as a professionally directed effort to insure that care provided to patients is of high quality, appropriate duration and rendered in an appropriate setting at reasonable cost.

Practice Evaluation:

The statement which was adopted by the Reference Committee reads as follows: "The American Medical Association encourages increased use of office or hospital outpatient facilities and use of these facilities for diagnostic testing prior to hospitalization wherever medically feasible and where quality of services can be assured."

Federal Trade Commission Interference:

There was considerable discussion and the following Resolution was presented: "RESOLVED, that the American Medical Association seek legislation which will permit constituent medical societies of the AMA to review the reasonableness of physicians' charges to protect the public, and BE IT FURTHER RESOLVED that the American Medical Association seek legislation if necessary to protect the role of constituent component medical societies in the discipline of members who charge excessive fees or exploit their patients."

Voluntary Cost Containment Measure:

The report is as follows in a Resolution from the Reference Committee: "RESOLVED, that the AMA continue to recognize that a structured voluntary cost containment program will meet the objectives of reducing the rate of escalation of health care costs without the inflationary effects of further government regulations, and BE IT FURTHER RESOLVED that any cost containment program should not withhold necessary medical care or unduly restrict medical research."

Government Regulations in Cost Containment:

The Committee considered this and resolved that the AMA should continue to urge the federal government to assess the impact of its regulatory systems on the cost of health care and to eliminate overlapping and unnecessary regulations, and further resolved that the AMA continue its present efforts to inform the medical profession and the public of that portion of increased health care costs attributable to such regulatory systems.

HMO Promotion under Medicare:

This was discussed by the committee and the following statement was made: "The Resolution called for the House of Delegates to condemn the use of public funds to promote enrollment of Medicare beneficiaries in HMOs over other forms of health care delivery." The Resolution correctly identified the inappropriateness of the use of federal funds to promote one type of health care delivery system over other forms, and we are pleased to note that the intent of this Resolution is already being implemented through communicaton from AMA to the Department of Health, Education and Welfare.

Patient Medical Records:

Resolves were made to continue AMA efforts to insure privacy of medical records and to continue to favor state rather than federal legislation on the subject. □

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Brief Summary

INDICATIONS: For the prevention and treatment of nocturnal recumbency leg muscle cramps, including those associated with arthritis, diabetes, varicose veins, thrombophlebitis, arteriosclerosis, and static foot deformities.

CONTRAINDICATIONS: Because of the quinine content, Quinamm is contraindicated in women of childbearing potential, in pregnancy, in patients with known quinine sensitivity, and in patients with glucose-6-phosphate dehydrogenase deficiency. Hemolysis (with the potential for hemolytic anemia) has been associated with a G-6-PD deficiency in patients taking quinine.

PRECAUTIONS: Thrombocytopenic purpura may follow the administration of quinine in highly sensitive patients. Recovery will follow withdrawal of the medication.
Cinchona alkaloids, including quinine, have the potential to depress the hepatic enzyme system that synthesizes the vitamin K-dependent factors. The resulting hypoprothrombinemic effect may enhance the action of warfarin and other oral anticoagulants.

ADVERSE REACTIONS: Aminophylline may produce intestinal cramps in some instances, and quinine may produce symptoms of cinchonism, such as tinnitus, dizziness, and gastrointestinal disturbance. if ringing in the ears, deafness, skin rash, or visual disturbances occur, the drug should be discontinued.

DOSAGE AND ADMINISTRATION:
1 tablet upon retiring. When necessary, 1 additional tablet may be taken following the evening meal.

Product Information as of September, 1977

U.S. Patent 2,985,558

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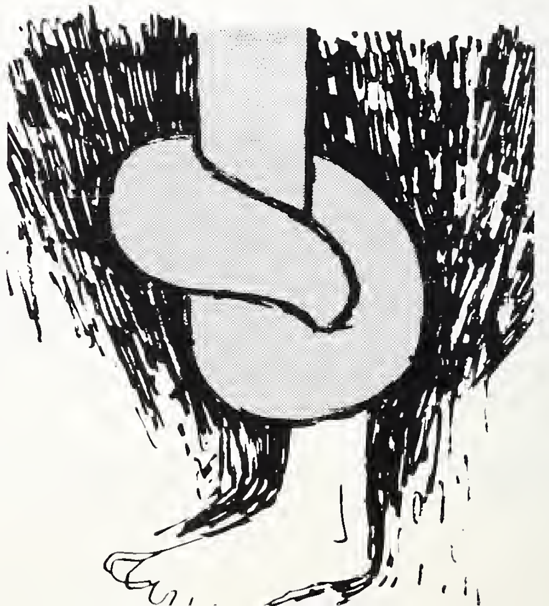

Editorials

PHYSICIAN-PATIENT AGREEMENTS AND EXPECTATIONS: A LEGAL PERSPECTIVE

Dr. Van Camerik, in his article in this issue of *The Journal,* asserts that patients frequently hold unrealistic expectations concerning the results of medical treatment. He further asserts that patients' inappropriate expectations result in many unfounded medical malpractice actions against physicians. In his essay, Dr. Van Camerik contends that the risk of malpractice litigation can be reduced radically if prior to commencing treatment the physician and patient execute a formal statement of shared expectations. Dr. Van Camerik asserts that if a physician informs a patient of the reasonable expectations of a treatment and the patient agrees to accept these reasonable expectations, the physician will substantially reduce the risk of patient disappointment and resulting malpractice litigation. In essence, Dr. Van Camerik appears to view the statement of shared expectations as a method of insulating a physician from unfounded malpractice actions.

In this editorial comment I will analyze Dr. Van Camerik's proposal from a legal perspective. More specifically, I will attempt to analyze the extent to which the written statement of shared expectations proposed by Dr. Camerik could affect a physician's liability in a malpractice action actually commenced against him. In addition, I will suggest modifications to Dr. Van Camerik's proposal designed to limit a physician's potential liability in actual malpractice litigation.

From a legal perspective the most significant feature of Dr. Van Camerik's proposal is that the statement of shared expectations may constitute a contract between the physician and the patient under which the physician promises to achieve a specified result, the shared expectation. In legal terms the statement of shared expectations may constitute an express warranty that the physician will achieve the specified result. If the statement of shared expectations is viewed as an express warranty, the statement will greatly expand the scope of the physician's liability to a patient.

Normally, in order to hold a physician liable in a malpractice action, the patient must establish that the physician was negligent. In such an action a showing that the treatment harmed the patient is not sufficient to impose liability upon the physician. In addition to establishing that the treatment proximately caused harm, the patient must establish that the physician failed to observe the appropriate standard of care in treating the patient. In other words, in a malpractice action sounding in negligence, a physician cannot be held liable to a patient for harm caused the patient if in treating the patient the physician observed the appropriate standard of care.

In contrast, when a physician expressly warrants that a treatment will achieve a specified result and the treatment fails to achieve the result, the physician will be liable to the patient for failure to achieve the warranted result, even though the physician complied with the highest standard of care in treating the patient. For example, in *Hawkins v. McGee,*[1] a case that has gained popular notoriety because of its prominence in the *The Paper Chase,* a physician was held liable for breach of an express warranty to make a patient's hand a hundred percent perfect despite a finding that the physician was not negligent in treating the patient. In recent years some courts have upheld findings that a warranty of cure was created on very scant evidence.[2] Unless carefully worded it is not improbable that the statement of shared expectations proposed by Dr. Van Camerik would be viewed as an express warranty, thereby expanding the scope of the physician's liability beyond negligence. A further reason for caution in implementing Dr.

Van Camerik's proposal is that it is probable that a physician's malpractice insurance policy will not cover liabilities resulting from breach of warranty.[3] The increased risks arising from the warranty theory of liability do not dictate a conclusion that Dr. Van Camerik's proposal should be abandoned. Rather, the risks indicate that the statement of shared expectations should be carefully drafted to preclude an interpretation that the physician is warranting that the treatment will achieve the expected result. In order to protect himself against the risks of warranty liability a physician might be well advised to consult his attorney and malpractice insurance carrier before implementing a statement of shared expectations procedure.

A second legal issue raised by Dr. Van Camerik's proposal is the possibility that a physician may use the statement of shared expectations to narrow the scope of his malpractice liability for negligence. A physician can be held liable for negligent malpractice only if in treating the patient the physician failed to observe the appropriate standard of care. Traditionally courts have defined the applicable standard of care as a matter of law. Recently, however, it has been suggested that the applicable standard of care should be viewed as a matter of agreement between the physician and patient.[4] The proposed statement of shared expectations could be used as a vehicle for establishing the standard of care by agreement.

The desirability of establishing the standard of care by agreement can be appreciated by focusing upon a case that highlights judicial evolution in defining the standard of care as a matter of law. Traditionally the standard of care imposed by courts was the standard customarily observed by physicians practicing in the same locality as the defendant. Recently, however, courts have exhibited a trend to abandon the "locality rule" in favor of a national standard of care.[5] A more disturbing development is illustrated by the 1974 decision of the Washington Supreme Court in *Helling v. Carey.*[6] In *Helling* the court held an ophthalmologist liable for medical malpractice as a matter of law for failing to administer a "pressure test" for glaucoma upon a 32-year old patient, although the defendant had established that the uniform practice in the medical profession was not to administer the pressure test to patients under 40 years of age. A physician seeking to avoid the risks graphically illustrated by *Helling v. Carey* may be able to define by prior agreement the standard of care that he will be obligated to observe, and thus preclude a court from imposing a higher standard of care after the fact. Although courts would certainly refuse to enforce an agreement that purports to hold a physician to an unconscionably low standard of care, a court should enforce a term that limits the physician's obligation to compliance with the standard of care customarily observed by the profession in the physician's locale. It should be noted, however, that a court may view the terms defining the standard of care as an attempt to exculpate the physician from liability for negligence and refuse to enforce the terms as a matter of public policy. Nevertheless, a physician may be able to use the statement of shared expectations to limit potential liability by including a provision in the statement expressly defining the standard of care the physician is obligated to observe during the course of treatment.

In conclusion, Dr. Van Camerik proposes the use of a physician-patient agreement to define a patient's reasonable expectations concerning a course of treatment. By dispelling a patient's unrealistic expectations prior to treatment, the physician can avoid malpractice suits that result from unnecessary patient disappointment. Dr. Van Camerik does not, however, explore the possibility that the statement of shared expectations may be used to define the scope of a physician's legal obligation. In this editorial I have commented briefly upon two aspects of a physician's liability that the statement of shared expectations may affect. First, unless carefully drafted the statement may constitute an express warranty and expand the scope of the physician's potential liability. Second, the physician may be able to use the statement to define by agreement the applicable standard of care and thereby narrow the scope of his potential liability in negligence malpractice actions. ☐

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ERRATUM

In the article, "Does MCAT Predict Clinical Performance," in the August 1979 issue of *The Journal*, Louis A. Marco, M.D., should have been listed as a co-author. Our apology to Dr. Marco.

ARE STATE MEDICAL JOURNALS OBSOLETE? A REBUTTAL

A "professional brief" in the August issue of *Medical Economics* asks, "Are state medical journals obsolete?" Cited is a study conducted of the authorship of articles in *The Journal of the South Carolina Medical Association*. As is so often the case with excerpts, only one side of the issue was presented. Some explanation, then, is needed.

Our findings were summarized at the 1978 SCMA annual meeting. In the course of this century, there has been a perceptible decline in the percentage of articles in *The Journal* written by *practicing physicians*. This decline is especially apparent in the smaller cities and towns. It was suggested that growth of medical school faculties may have inhibited practicing physicians from writing.

The other side of the issue, also emphasized in our study, was that there is a definite *need* for scholarly self-expression by practicing physicians. Throughout the years, eloquent expressions of this need have appeared in *The Journal*. A major reason for the continued existence of state medical journals is to provide a place for practicing physicians to publish their findings or their thoughts and philosophies.

We continue to encourage manuscripts from practicing physicians. We also feel that there is a need for more *collaboration* between practicing physicians and medical school faculty members. Computer technology may be of great benefit to such collaborative efforts. Recently, one of our officers has suggested that an award be sought for the best scholarly effort by a practicing physician published each year in *The Journal of the South Carolina Medical Association*. Hopefully, this creative suggestion will enhance, even further, our viability. Meanwhile, it is refreshing to note that all four scientific articles in the current issue are by physicians who are not identified as full-time faculty members.

— CSB

COST CONTAINMENT AND PHYSICIANS' FEES: THE ST. PETER'S PARISH SOLUTION

What is past is prologue, goes the famous aphorism carved in the entrance way to the National Archives in Washington. In these days of heated discussion and of talk of hard times ahead, it might be of interest to reflect on a solution offered by a citizens' committee of St. Peter's Parish, located about 75 miles south of Charleston, one hundred and thirty-five years ago. This reflection was offered by Dr. Joseph I. Waring, and published posthumously.[1]

Although we often think of the 1830's and 1840's as times of great prosperity in South Carolina, this was generally not the case. Resources seemed impoverished, the price of cotton was falling, and many South Carolinians had departed for the flourishing West. A Senator wrote of Charleston and of the South Carolina low country: "Our merchants are bankrupt or driven away . . . our mechanics in despair; the very grass growing in our streets, and houses falling into ruins; real estate reduced to one-third part of its value . . . Fields abandoned . . . agriculture drooping . . . the planter striving with unavailing efforts to avert the ruin which is before him."[2] Against this background, a public meeting was held in St. Peter's Parish to discuss the subject of the cost of medical care.

Not surprisingly, a committee was appointed. The committee came back with some stern recommendations. Reflecting on physician's fees, they gave this opinion:

> "These charges . . . are entirely disproportioned to the present hard times. Indeed, the great disparity between the price of medical service and the value of the services rendered, is so notorious, that it is a matter of surprise that the attention of the agricultural classes has not long before this been called to the subject, as a source which unjustly absorbs so large a portion of their *hard earned* incomes. Your Committee further report, that they can find no uniformity in the medical charges in this Parish, and they recommend to you to adopt and to have published the subjoined fee bill. . . ."

An excerpt from the fee bill is shown in the accompanying table. According to Dr. Waring, the outcome of this proposed solution is unknown. Nevertheless, Dr. Waring concluded carefully: "Perhaps we need a new voice from St. Peter's Parish to guide our government councils today."

— CSB

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TABLE

REPRESENTATIVE FEES, FROM THE COMMUNITY FEE BILL FOR PHYSICIANS, ST. PETER'S PARISH, 1844

Service	*Fee*
Delivery (uncomplicated)	$3.00
Delivery (complicated)	6.00
Gonorrhea (cure)	2.00
Syphilis	4.00
Amputation of a limb	5.00
Setting a simple fracture	2.00
Setting a compound fracture	5.00
Examining a case without prescribing medication	0.25
Attention, detention, or bedside attendance, per hour	0.25
Operation for cataract	5.00
Opening an abscess	0.25
Attendance at court as a witness, per day	1.00

President's Page

MEMBERSHIP — THE FUTURE

When the South Carolina Medical Association was chartered in 1848, it established three primary objectives: *supression of quackery, promotion of high-toned ethical standards for members of the profession in the state, and the elevation of the qualifications for graduation in the South Carolina medical schools.* These objectives in terms of what were expected in that day are being fulfilled, but the work of the association continues and expands at an increased rate. The extra problems facing us today are problems common to all of us (academic and practicing physicians), brought about by major political and social events which bring into play third party payers and governmental agencies. We must take advantage of the increasing realization that there is a need for an umbrella organization — united, working in concert with specialty societies and academe for the common good. With planning now, our growth and success can be assured.

Where will our growth potential be found?

Our physician population can be divided into two general groups. Sixty percent of our active, non-federal physicians are in private offices. The other 40 percent are residents (17 percent) and physicians who are hospital based, college or university based, in teaching, administration or research. In January, 1979, South Carolina had a total of 3,343 active physicians (2,097 office based), with a gain of 523 physicians and loss of 315. In 1978, we experienced a net gain of 208 physicians in South Carolina which compares to the net gain in 1977 of 164 physicians. Each of these years reflects a favorable in-migration of physicians.* This in-migration has averaged around 300 physicians over and above those who graduated from the Medical University of South Carolina and stayed in the state to practice.

The 40 percent is expected to grow as we continue the 165 at MUSC and increase the enrollment of medical students to 100 at USC, with residency programs sufficient to offer a slot for each graduate, plus the necessary full-time faculty for medical schools and major hospital teaching programs. By 1983, the year that the pipeline will be full, a conservative estimate of personnel involved would reach 2,300 (1,060 students, 795 residents and a minimum of 500 full-time faculty members). Under the direction of our President-Elect, Dr. Halsted M. Stone, and Ad Hoc Committee on Membership is working to contact physicians who are not members. In addition, officers and councilors are visiting medical societies to explain our activities and to obtain first-hand input from the society members. We welcome invitations to meet with you.

We are making progress. We are growing, but we are at the beginning of a great influx of new physicians in the state and an increase in the number of full-time faculty. We must be prepared to take advantage of this new opportunity by obtaining new members and to direct the changes that are inevitable. For too long there has been too much talk about "Town and Gown" strife. With common problems, we grow closer. We will solve problems better by working together, and there is every indication that there is a willingness to do so.

We have now reached an all-time high in total membership of 2,161. Would 3,000 members in SCMA by 1983 be asking too much?

Harrison L. Peeples, M.D.,
President

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* S. C. Health Cooperative Statistics System statistics.

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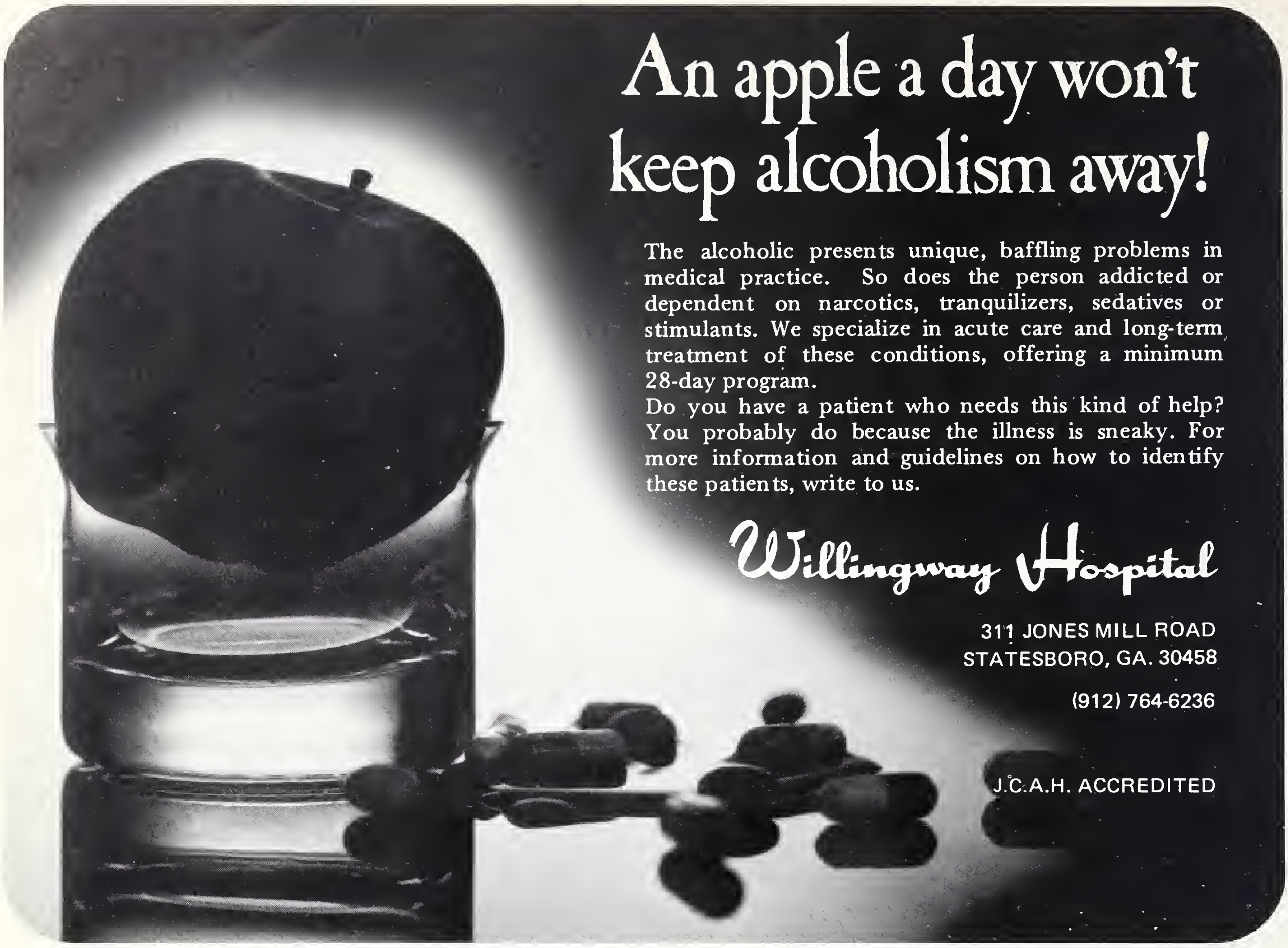

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SOUTH CAROLINA MEDICAL ASSOCIATION

AUXILIARY

AUXILIARY CONVENTION — 1979

The fifty-sixth annual convention of the AMA Auxiliary convened in Chicago July 22-25 at the Drake Hotel. The AMA convention was held at the Marriott Hotel.

South Carolina was well represented by our board members: Billie Brady of Greenville; immediate Past-President, Nellie Claire Brown of Rock Hill; President, Marge Adams of Columbia-Easley; President-Elect, Karin LaBruce of Charleston; Lib Peeples of Estill; and Jennie Tanner of Columbia. State AMA-Chairman, Carol Harter of Anderson, was also in attendance as were Skippy Adkins and Mary Ivester of Charleston.

Among those items voted on was the proposed dues increase which did pass. A preview of the National Campaign for Nutrition and Physical Fitness, "Shape Up for Life," was presented.

Mrs. Bergnes told the group that "Unity is vital to the strength of our organization."

Delegates to 1979 AMA Auxiliary Convention from South Carolina. left to right: Mrs. Wayne C. Brady, Mrs. Alton G. Brown, Mrs. James F. Adams, Mrs. Arthur LaBruce. Not pictured, Mrs. Harrison Peeples.

Reception honoring Mrs. Manuel Bergnes, AMA Auxiliary President. left to right: Mrs. Wayne C. Brady, Mrs. Arthur LaBruce, distinguished speaker, Edwin Newman, and Mrs. Alton G. Brown.

Shown with the Membership Award is Mrs. Arthur LaBruce (SCMA Auxiliary President-Elect), Mrs. Alton G. Brown (Past SCMA Auxiliary President), and Mrs. James F. Adams (SCMA Auxiliary President).

Shown from left to right are Mrs. James F. Adams, Dr. Harrison Peeples (President SCMA) and Mrs. Alton G. Brown.

S. C. Delegates present at the House of Delegates meeting are: Mrs. Alton G. Brown, Mrs. Arthur LaBruce, Mrs. James F. Adams, Mrs. Waitus Tanner.

Mrs. Wayne C. Brady (Board of Directors of AMA Auxiliary) is pictured with Mrs. Manuel Bergnes (President AMA Auxiliary) at a reception honoring Mrs. Bergnes.

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THE JOURNAL
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INTRODUCTION

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These proceedings represent the report of the Second National Conference on Emotional Stress and Heart Disease held at Hilton Head Island, South Carolina, October 13-15, 1978. This conference was a follow-up to the First National Conference held at Myrtle Beach, South Carolina, October 1-4, 1975, the proceedings of which were published as a supplement to *The Journal of the South Carolina Medical Association* in February, 1976. The interest generated in the subject is exemplified by the fact that 3500 copies of those proceedings have been distributed to 45 states and 20 foreign countries.

It was felt that the first conference had presented substantial data to indicate that emotional stress is a cardiovascular risk factor, established a working definition applicable to clinical use, identified criteria for its detection and measurement, and established guidelines for intervention in a preventive and therapeutic manner.

The Task Force on Emotional Stress and Heart Disease of the South Carolina Affiliate of the American Heart Association organized this conference to ascertain progress in the field during the three year interim. The Second Conference brought together a multi-disciplinary group of national leaders in the field to continue exploration and dissemination of information in this complex area. Their findings are presented in these proceedings.

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STRESS AND CARDIOVASCULAR DISEASE*

JAMES C. BUELL, M.D.**
ROBERT S. ELIOT, M.D.***

The current interest in the relationship between stress and cardiovascular disease stems from a sizeable body of evidence implicating psychosocial conflict, emotions and patterns of behavior in the pathogenesis of cardiovascular disease, arrhythmias and sudden death. This interest has been heightened by observations that the traditional risk factors of elevated blood cholesterol, hypertension, and cigarette smoking are absent in more than half the new cases of coronary heart disease encountered in clinical practice.[1] In view of our incomplete knowledge concerning the pathogenesis of coronary heart disease, it is not surprising that preventive measures confined to a few factors have met with limited success.[2] Nevertheless, consistent and persuasive findings are recurrently reported linking coronary and hypertensive heart disease to prolonged emotional stress, behavioral patterns, sociocultural mobility and changing life events. Atherosclerotic and hypertensive cardiovascular diseases appear to be epidemic afflictions of twentieth century industrialized societies, and the prevalence of these conditions parallels the increasing complexity of the social order. For these reasons, stress is justifiably the subject of much current interest and investigation, although to date, the field remains controversial with confusing terminology and imperfect methodology.

HISTORICAL BACKGROUND: DEFINING "STRESS"

Professor Hans Selye developed and popularized the term "stress" while describing the general adaptation syndrome in 1946[3]; but Walter Cannon had first used the term in a physiological sense in 1914.[4] In addition to developing the fight or flight concept, Cannon also described the use of an engineering concept of stress and strain in a physiologic context.[5] He clearly conceived of stress as involving both physical and emotional stimuli, his principal thesis being that "The chance of gaining insight into the strength and endurance of stabilizing factors of the organism, and thus its ability to resist the operation of disturbing forces, makes it worthwhile to inquire where the limits lie beyond which stresses overwhelm these corrective factors and significantly alter the steady state of the internal environment."[5] Later, Wolff[6] described stress as physical, psychologic and symbolic stimuli, all of which are capable of eliciting physiologic responses.

Today, stress is more commonly used in a non-physical sense. A contemporary definition was offered recently at the National Conference on Emotional Stress and Heart Disease: "an obviously painful or adverse force which induces distress or strain upon both the emotional and physical make-up."[7] Appley and Trumbull[8] point out that as a psychological concept, stress has been used not only to refer to extreme environmental or psychosocial conditions, but also as a substitute for what others might call anxiety, conflict, ego threat, frustration, or threat to security. Lazarus[9] has suggested that we view psychological stress more broadly, including not only stimuli and response but also the intervening psychological experimental factors which ultimately determine stimulus response relationships.

Thus, it is apparent that the term stress affords little understanding. It means stimulus to some, response to some, interaction to others, and complex combinations of these to still other workers. Indeed, it has even been proposed that the most desirable course would be to use the term stress sparingly, if at all, and to adhere to terms for stimulus and response parameters that are operationally sound and descriptive.[10] Ob-

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served pathophysiologic responses often correlate well with the inability of the individual to adapt harmoniously to the demands of twentieth century industrialized societies. Eliot has termed this twentieth century phenomenon the "state of invisible entrapment," wherein the alarm reaction maintains a chronic state of visceral vascular readiness and few, if any, personally acceptable options exist for prompt resolution of the dilemma.[11]

PSYCHOSOCIAL STRESS IN ANIMALS

Whereas demographic evidence indicating stress in the pathogenesis of human cardiovascular disease remains circumstantial, data from experimental animals are direct and unmistakably link psychosocial disruption to pathological changes in the cardiovascular system. Work summarized by Mason[12] has shown unequivocally that psychosocial stimuli can elicit either of two neuroendocrine responses, one involving arousal of the pituitary-adrenal cortical system and the other that of the sympathetic-adrenal medullary system. In general, social interactions leading to downward displacement in the hierarchy lead to stimulation of the pituitary-adrenal cortical system, with mental depression, decreased gonadotropin levels, enhanced vagal activity, gluconeogenesis and pepsin production. The sympathetic-adrenal medullary system is called into play as agonistic behavior is invoked in an attempt to maintain status and prevent threatened loss of esteem and/or related objects of attachment.[13] Numerous animal studies have demonstrated that adverse psychosocial stimulation will result in pathological disturbances leading to frank disease states.[14, 15, 16]

ACUTE PSYCHOSOCIAL STIMULATION

Corley and co-workers[17] studied 21 restrained squirrel monkeys. After approximately a week of eight hours on and eight hours off electrical-shock avoidance, they found a significant incidence of acute myocardial degenerative lesions. Control animals were free of histologic changes. Ten animals that were only restrained during the period of study showed minor ST segment changes. All the animals exposed to shock avoidance plus restraint, however, showed marked ST segment changes with arrhythmias and extensive myocardial fibrosis. The authors concluded from this study that restraint combined with shock-avoidance testing induced autonomic disturbances resulting in significant myocardial necrosis.

In the realm of hypertension, Von Holst[18] performed detailed experiments in adult male tree shrews in which a subordinate animal was introduced to another male that was an experienced fighter. The dominant animal immediately attacked while the intruder submitted. The two were separated before injury could occur, but the subordinate was placed so that he could see the dominant animal without being attacked by him. The emotionally aroused subordinate lay still, watching the other's movement, more than 90 percent of his waking time. His tail hair remained erect, indicating a sustained sympathetic arousal. Within two to 16 days, despite eating and drinking, the subordinate animal would fall into coma and die in association with a rising blood urea nitrogen and histologic evidence of renal insufficiency, pointing to an acute renal vascular change as the cause of death. Hoff et al.[19] observed development of renal cortical necrosis following stimulation of the frontal cortex of the cat. Knapp et al.[20] have demonstrated that continuous infusion of norepinephrine into the renal artery of the dog for two hours will lead to irreversible renal failure.

The dichotomy of response patterns to a stressor is illustrated in the report of Corley, Mauck and Shiel.[21] They confined pairs of squirrel monkeys to chairs for eight hours a day, during which time one animal had to turn off a light once a minute or both received a tail shock. The avoidance animal showed evidence of sympathetic arousal by hypertension of 150/110 mm Hg and eventually developed myocardial fibrosis and degeneration. It did, however, maintain physical activity, outlasting the helpless member of the pair which could neither control the situation nor cope with it. Five of the six helpless monkeys collapsed with bradycardia and four died in asystole. Thus, the effects of stimulation were manifested in two different response patterns, depending on which component of the neuroendocrine system was dominant. Nevertheless, acute experiments do not last sufficiently long to permit examination of the gradual deterioration and slower transformation from health to incapacity that are more characteristic of clinical cardiovascular disease.

CHRONIC PSYCHOSOCIAL STIMULATION

Reports by Lapin and Cherkovich[22] were among the first successful demonstrations of the pathogenic potential of sustained agonistic social interaction. Their work involved changes in the social situation of dominant Hamadryas male baboons. The male of this particular species adopts its females when they are still immature and develops an intense attachment for them. When the dominant male was separated from the females and his young, and another male was put ino the females' cage in full view of the former consort, the displaced male showed intense agitation. After several months, hypertension and other evidence of chronic cardiovascular disease developed. While some of the studies were aimed at developing experimental neuroses rather than deliberate disease, the authors encountered 16 cases of hypertension, 19 cases of coronary insufficiency, and six cases of acute myocardial infarction.

Ratcliffe[23] observed that deaths in animals and birds from arteriosclerotic coronary heart disease increased from less than one percent in 1948 to more than 10 percent during 1968. Diet and mean age at death were no different, but he noted that 1948 was the start of the zoo's attempt to assemble family groups. This contrived social grouping resulted in conflicts, breeding failures, and abnormal behavior within the "family groups." He eventually concluded that the increase in coronary heart disease resulted from behavioral responses to social situations.

In an experimental follow-up to this observation, Ratcliffe et al.[24] studied psychological responses of swine to separation occurring after the primary social bonds of grouped animals had been formed. At the end of a year, the separated animals showed a significantly greater incidence, extent, and severity of coronary atherosclerosis. Whereas the grouped and paired swine responded to human visitors with friendly grunts and squeals for a handout, the separated swine, especially the normally friendly females, failed to respond to visitors, even refusing offers of added corn. The results of this quantitative study suggested that a sustained emotional disturbance was significantly associated with chronic pathophysiological changes.

Weber and VanderWalt[25] produced cardiomyopathy in New Zealand white rabbits by alternately housing them singly and then crowding them again. In the course of 10 months, 35 of the 48 animals had expired, 10 dying during the first week, 10 more during the first month, and the rest at intervals during the succeeding months. In those that died within a few days, severe myocardial necrosis and interstitial edema with patchy accumulations of acid mucopolysaccharides were observed in the subendocardium and myocardium. Those that survived longer developed foci of myocardial fibrosis with collagen fibers and endocardial fibroelastosis; there was compensatory myocardial hypertrophy in the fibers adjacent to the fibrotic areas.

The ethological approach seems to indicate that at least two identified neuroendocrine responses are evoked and both may have pathological consequences, as discussed by Raab[26] and Eliot.[27] These responses are mediated on the effective integrative level through two mechanisms: (1) the limbic striatal system, which is involved with social, emotional, and self-preservation behavior; and (2) the brain stem-hypothalamic system, which is concerned with sex, hunger, thirst, and sleep drives. It is in the recruitment of these mechanisms at the cognitive, integrative level that species differences become most manifest. This involves the frontal association cortex and the parietal and temporal cross-modal association areas, termed the sociocultural brain. It is only in humans that these structures attain overwhelming proportions compared with the rest of the organ.

PSYCHOLOGICAL AND SOCIAL STRESS IN HUMANS

The observation that the head influences the heart in humans is certainly not a new concept. Almost 2,000 years ago, Celsus recognized that emotional states could influence the heart when he commented that "bathing, exercise, fear and anger and any other state of the mind may often be apt to excite the pulse."[28] In 1628, William Harvey reaffirmed the observation by stating that "every affection of the mind that is attended with either pain or pleasure, hope or fear, is the cause of an agitation whose influence extends to the heart."[29] In more recent times, it has been implicitly recognized from Hunter's[30] prediction of the circumstances of his death, to Osler's[31] descriptions of the emotional influences surrounding angina pectoris, to Cannon's observations on voodoo death,[32] that the human psyche and circulation are somehow interrelated.

The clinical observations associating psychosocial factors and stress with cardiovascular disease, arrhythmiagenesis, and sudden death are so extensive that they cannot be adequately covered by this discussion. The reader is referred to a number of comprehensive reviews in the literature.[33, 34, 35, 36] Here, we will briefly review the data on socioeconomic factors, life-change indices and behavior patterns.

SOCIOECONOMIC FACTORS

The highest coronary disease rates appear to be found in Finland and the United States, with the lowest rates in Greece, Yugoslavia and Japan. Occupation and marital status inconsistently correlate with coronary disease. However, the associated way of life for an occupation appears to contribute to coronary risk above and beyond the physical labor involved in the job itself. Furthermore, the data on social mobility and status incongruity may be valid predictors only in certain places or at certain times, only for certain presentations of disease, or only when other variables are present.

The limitations of such demographic approaches are well illustrated in the studies of the incidence of cardiovascular disease and sudden death at the Kennedy Space Center during the mid 1960's.[37] The cardiac death rate among young men at the Kennedy Space Center was nearly 50 percent higher than that of age and sex matched control groups. The divorce rate was three of every four marriages, and this microcosm led the nation in per capita consumption of alcohol. Despite what might have been expected, the most significant factors were: 1) the perceived national urgency of putting a man on the moon by the end of 1969; 2) the great national and international visibility of the Center, in which a dramatic failure would have adverse occupational consequences; 3) the problems of interpreting and coordinating the work of approximately 300 different major industrial subcontractors, particularly for administrators and managerial personnel engaged in interorganization liaison; 4) the progressive decline in budget and employment; and 5) the threat of loss of employment and income from there being no demand for overspecialized, highly trained skills in the common marketplace. In short, the project demanded an ever-increasing, if not frenzied, pace of production with the ultimate reward of nearly inevitable dismissal.

This investigation revealed that both occupational and age factors were highly associated with psychoneuroticism and that there was a remarkable incidence of abnormal resting electrocardiograms. The highest incidences of psychoneuroticism were seen among foremen, managers, administrators, and unskilled manual laborers, who also demonstrated the highest percentage of abnormal resting ECGs.[38] In every instance, the scores of psychoneuroticism observed in groups at Cape Kennedy were higher than those of a matched and controlled sample of respondents from a major epidemiological study of social psychiatric impairment.

LIFE PROBLEMS, DISSATISFACTIONS AND CHANGE

There is also considerable evidence suggesting that coronary disease is often associated with anxiety, depression, psychophysiological complaints, sleep disturbances, fatigue and emotional drain. This family of variables is perhaps more highly associated with angina pectoris than myocardial infarction. Whereas investigations regarding sleep disturbances are limited, associations with cardiovascular disease are a highly consistent finding. It is reasonable to suspect that these spheres of investigation might be highly interrelated, yielding similar correlations because of a common denominator. The pivotal point of this common denominator might well be the ability of the individual to cope with life changes and dissatisfactions from the perspective of personal success or failure in dealing with his environment.

The association between life-change indices and myocardial infarction remains provocative, but there is a stronger association between cardiovascular disease and anxiety or depression than with occupation or other demographic data. This suggests that environmental provocation cannot be viewed in isolation. The multitude of adaptive resources and mechanisms at the disposal of an individual and his or her subjective interpretation of their suitability to outcome are probably equally important factors.

CORONARY-PRONE BEHAVIOR PATTERN

The concept of coronary prone behavior pattern is not new. Even before the turn of the century, Osler described the typical patient with coronary disease as "a keen and ambitious man, the indicator of whose engine is always set at full speed ahead."[39] This coronary-prone behavior pattern characterized by Rosenman and Friedman[40] as type A, is manifested by some or all of the following: intense striving for achievement; competitiveness; being easily provoked; impatience; time urgency; abruptness of gestures and speech; overcommitment to vocation or profession; excesses of drive and hostility. Russek and Russek[41] claim that restlessness during leisure hours and a sense of guilt during periods of relaxation are among the most characteristic traits of the youthful coronary patient.

Although a number of studies have associated type A behavior with risk of coronary disease, restricted views and poor definitions of variables pose definite problems with regard to the coronary-prone behavior pattern.[42] The initial studies of such behavior were derived from a structured interview designed to elicit subject responses with particular emphasis placed on motor characteristics. Using different scales in their interviews, a group of investigators showed ratings of the responses in agreement varying between 64% and 84%. Besides the concern for reliability, critics present other arguments regarding the interview method of assessment. First, the criteria for scoring are nowhere specifically spelled out, necessitating personal instruction by Rosenman, Friedman, or one of their proteges to initiate the technique. Second, wide individual differences have been observed in the ability of investigators to gain intuitive practical skill in the assessment of behavior patterns.

Considering that the initial classification relied heavily on motor and hostility characteristics, it is not surprising that the self-administered, computer-scored questionnaire approach of the Jenkins Activity Survey is less predictive. Critics of the test further suggest that it fails to examine the hostility quotient and the facility with which an individual can be aroused, both of which are viewed as fundamental to the type A behavior pattern by Rosenman and Friedman. When the Jenkins Activity Survey scores of men who had suffered coronary heart disease at least five years before were compared with those of a control group free of disease, it was found that they did not differ significantly on the A/B scale, the speed and impatience scale, or the job-involvement scale.

Caffrey,[43] in his studies of 25 monasteries, noted that those monasteries with a higher observed rate of coronary heart disease were consistently rated by judges as possessing an environment likely to foster the type A behavior. Further, while the mechanism of paternal influence remains unclear, correlations exist between type A behavior in fathers and in their sons.

The relationship between behavior pattern and serum cholesterol appears inconsistent. Dembroski and associates[44] studied type A and type B subjects engaged in reaction-time tasks and found significantly greater lability and magnitude of blood pressure and heart rate measurements in type A persons. Mean resting levels were not significantly different, but lability was greater in type A, suggesting greater lability of sympathetic-parasympathetic functioning. No difference between types was observed in either galvanic skin response or speed of reaction.

Jenkins[34] has emphasized that type A behavior is not the same as stress in that "it neither represents a stressful situation nor a distressed response, but rather a style of behavior with which some persons habitually respond to circumstances that arouse them."

We believe that future studies of stress and cardiovascular disease must accurately and clearly delineate the factors of stimulus, integration, and response, whatever terminology is employed. That psychosocial disruption is associated with a very high incidence of disease in an animal colony is as indicative for pathogenicity as the inoculation of a colony of test animals with a microbe. Relegating a particular bacterial species to the category of "pathogen" does not necessarily imply that the microorganism will invariably produce the same bodily responses or effects in all subjects or under all conditions in the same subject. Such a definition of pathogenicity, therefore, implies the element of potentiality.

Much as the interplay between inoculum dose and host resistance determines which individuals in a colony will develop disease, the magnitude of stress and the resiliency of the subject are opposing factors in determining the outcome in the interplay of stress and cardiovascular disease. One must seek the reasons for resiliency as well as for susceptibility, since those who flourish in contemporary society will probably be products of learned behavior. The only alternative is the biological time schedule of natural selection by extinction.

Physical scientists have long characterized materials by their ability to withstand applied force, their elastic properties and their stress-to-strain relationships. We believe such an approach may prove fruitful in attempting to unravel the dynamic interrelationships among humans, their emotions, and their environment in the pathogenesis of contemporary cardiovascular disease. ☐

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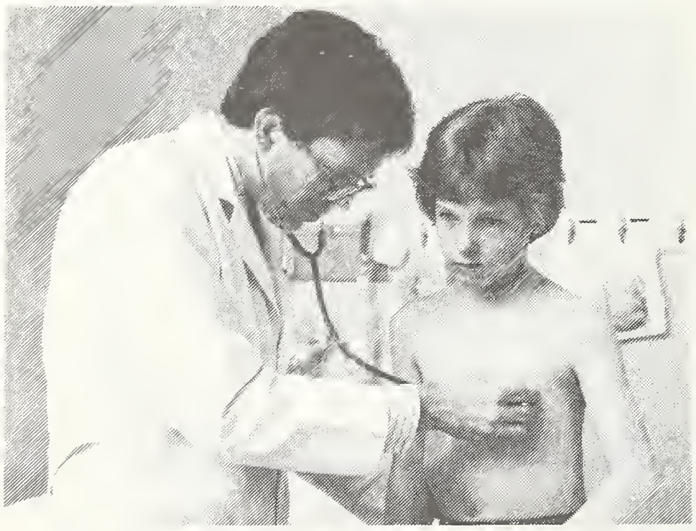

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PHYSIOLOGY OF EMOTIONAL STRESS: SPECIFIC RESPONSES

JAMES P. HENRY, M.D.*
DANIEL L. ELY, Ph. D.**

MASON'S COGNITIVE MEDIATOR THEORY OF STRESS

A discussion of the physiology specific for different types of emotional stress can well begin with John Mason's detailed differentiation between the arousal of the sympathetic adrenal-medullary system by the fight-flight response[1] which was first investigated by Cannon[2] and the pituitary adrenocortical response[3] with which the pioneer work of Selye[4] is associated. Mason's work has led away from concern with the nonspecific generalized adaptation response to various physical stressors and points to the importance of cognitive functioning.[5] He has recently described experiments showing that agents such as cold, heat, fasting, or exercise will fail to elicit either of the just mentioned neuroendocrine response patterns in monkeys if emotional arousal is carefully avoided. Noticing on the very first day an elevation of 17-hydroxycorticosterones (17-OHCS) steroids in monkeys that were deprived of food, while others received their ration, Mason and Muller (unpublished observations) realized that psychological as well as nutritional factors might be at work. They then carefully minimized novelty and uncertainty by allowing monkeys plenty of time for adaptation to experimental conditions and by permitting only familiar persons to handle them. Outside stimuli were eliminated by soundproofing the room and discomfort was minimized by giving monkeys unnourishing fruit-flavored pellets. In 11 experiments with eight monkeys, they found no change of steroids in association with fasting periods, although excretion of adrenaline increased and noradrenaline decreased.

Mason and others[5] were able to show that by ingeniously preventing emotional arousal in primates as well as in man they could put an animal through its paces without eliciting a 17-OHCS response. Thus by substituting the more natural act of climbing up and down a ladder repeatedly to get food instead of lifting heavy weights while being restrained in a chair, the subject's pitaitary adrenal-cortical system response was maintained at a low level. From this and other work, Mason and his associates concluded that conventional laboratory situations designed for the study of physical stressors such as immobility, exercise, heat, and cold, often elicited an appreciable degree of emotional disturbance due to discomfort or even pain and that these stimuli associated with the limbic system rank high among the most potent and prevalent sources of increased pituitary adrenocortical secretion.

Burchfield, Elich, and Woods[6] have recently shown that geophagia, the consumption of unnourishing dirt by a rat that is made to feel ill, will occur in response to any stressful occurrence; in fact, it is triggered by a purely emotional reaction to a perceived threat. Control rats exposed to distress signals from the experimental rats undergoing restraint and cold engaged in vigorous geophagia. Only the controls housed in another room were undisturbed. They cited Mason's viewpoint, mentioned previously, and interpreted their results as support for what they termed his cognitive mediator theory of stress. Mason's observations are critical because ironically, despite modern man's ability to control hunger, cold, heat, and hard physical labor, distress due to frustration and perception of various types of threats to well being and security remains. The psychosocial stimuli arising from a crying baby, an angry spouse, and a disgusted boss produce neuroendocrine responses of the greatest importance. We do not have to be starving, thirsty, wet or cold to experience stressful emotional arousal with activation of the limbic structures.

Social stimuli depend on one's perception of the social environment as mediated by personality variables, role, and status — it is these elements that arouse emotions. Some associate these variables with the right hemisphere, and Tucker and his associates[7] in their provocative article give evidence that there is greater right hemispheric than left hemispheric activation with emotional arousal in man.

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Modern man has to suppress strong impulses of anger and refrain from attacking someone who is frustrating him, and he must often cover up by pretending to be unafraid. It has been suggested that this is a new form of stress. The caveman or the animal, it is said, was able to express himself in a healthy fight-flight response, but this means of expression is not easily available to the hapless officeworker who must often inhibit his emotional response. Self-restraint, however, is required of all animals with a social hierarchy. Short shrift is the lot of socially deprived, formerly isolated monkeys that vent their irritation on an infant or a more dominant member.[8] A well adapted subordinate knows he must wait his turn patiently and show respect to those higher in the social hierarchy. This holds true not only for human hunter-gatherers but also for subhuman primates, carnivores, and rodents.[8] A monkey that has always been a member of a troop in which his parents were dominants is likely to attempt a dominant role and views the environment as a challenge. But an intruder from another troop which has been repeatedly defeated in attempting to become established is likely to perceive the new troop as unamenable to control and responds to threats with subordinated, passive behavior.[9]

FIGHT-FLIGHT AND DEPRESSIVE STRESS RESPONSES

It becomes of interest to contrast the physiological responses of the challenged dominant with those of the equally stressed subordinate. There is ample evidence of a special neuroendocrine response of the dominant to challenge, while a quite different set of hormonal and autonomic adjustments are characteristic of the subordinate. Behaviorally, he must often remain immobile as he watches the leaders and moves to get food and water only when safe to do so without reprisal. Figure 1 outlines these concepts.[10] If the psychosocial stimulus a mammal perceives is inhibited at higher levels by interaction with coping patterns, he will perceive it to be irrelevant in the context of his frame of reference. However, if he perceives it as relevant, there is arousal and his limbic system components will respond, initiating trains of responses which, as shown at the left and right of the diagram, differ for the challenged dominant and the defeated subordinate.

Right side of Figure 1: Recent work supports O'Keefe and Nadel's suggestion (as cited in O'Keefe and Dostrovsky[11]) that the limbic system's hippocampus functions as a cognitive mapping system. It provides information for place-learning by which the organism knows how to approach or how to avoid a particular location in the environment by an appropriate route. The

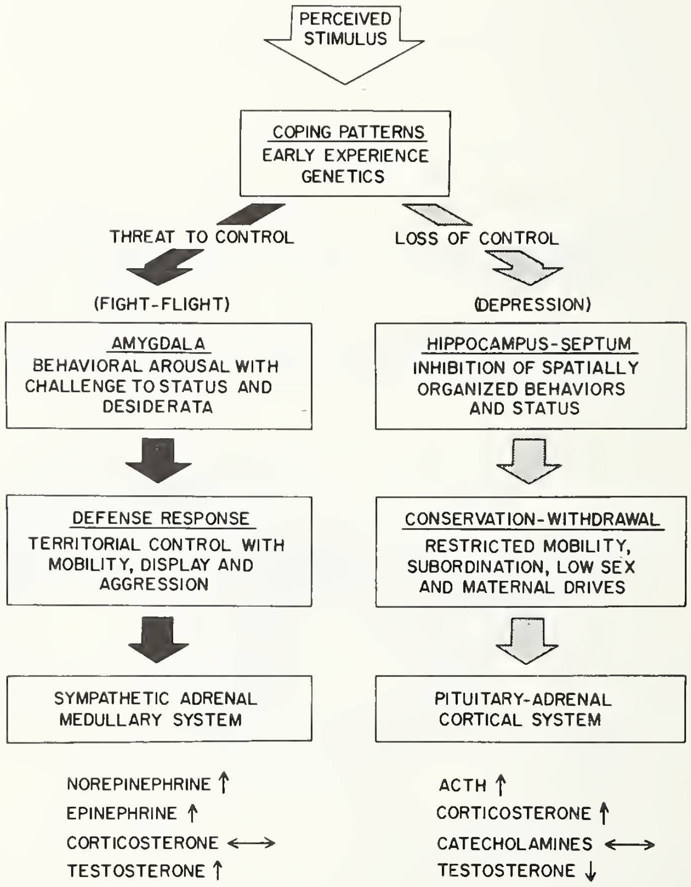
PERCEIVED STIMULUS
COPING PATTERNS
EARLY EXPERIENCE
GENETICS
THREAT TO CONTROL
LOSS OF CONTROL
(FIGHT-FLIGHT)
(DEPRESSION)
AMYGDALA
BEHAVIORAL AROUSAL WITH CHALLENGE TO STATUS AND DESIDERATA
HIPPOCAMPUS-SEPTUM
INHIBITION OF SPATIALLY ORGANIZED BEHAVIORS AND STATUS
DEFENSE RESPONSE
TERRITORIAL CONTROL WITH MOBILITY, DISPLAY AND AGGRESSION
CONSERVATION-WITHDRAWAL
RESTRICTED MOBILITY, SUBORDINATION, LOW SEX AND MATERNAL DRIVES
SYMPATHETIC ADRENAL MEDULLARY SYSTEM
PITUITARY-ADRENAL CORTICAL SYSTEM
NOREPINEPHRINE ↑
EPINEPHRINE ↑
CORTICOSTERONE ⟷
TESTOSTERONE ↑
ACTH ↑
CORTICOSTERONE ↑
CATECHOLAMINES ⟷
TESTOSTERONE ↓

FIGURE 1. If the psychosocial stimulus perceived by a mammal is not inhibited at higher levels by interaction with coping patterns and social assets, a response of the limbic system will take place. The amygdala and the sympathetic adrenal-medullary system are activated when the organism is challenged in its control of the environment. But when there is loss of control and failure to meet expectations, the hippocampal pituitary-adrenal-cortical system becomes more involved as the conservation-withdrawal response is aroused. The physiological consequences of these two response patterns differ as shown; the horizontal arrow indicates no change. (From J. P. Henry and P. M. Stevens: *Stress, Health, and the Social Environment*, Springer-Verlag, New York, 1977, Fig. 7-1, p. 119.) Reprinted with permission.

hippocampus appears to contain cells that locate a mammal's position in an environment by representing spatial arrangements.[12] There is evidence of a system of cells which signal a mismatch between the stored representation of the environment and a mammal's present perception. When this mismatch is perceived, exploratory activity is generated and the mammal ceases to practice old patterns, and under the influence of the adrenocorticotropic hormone (ACTH), he rapidly learns new information with which to update the cognitive map into a more suitable pattern for the new situation. Not only may this mapping function apply to location in physical space, but also to the concept of territory and home range in its broadest sense. Social organizations require their members to have an acute sense of status; they must "know their place" not only in the physical but also in the social hierarchic space.[10, 12]

An animal which, by becoming subordinate, loses social control may then be thought of as having lost hierarchic-social space. He does not control the symbolic territory of social relations. This helplessness may trigger hippocampal drives to the hypothalamus and from there to the pituitary adrenocortical mechanism.

Left side of Figure 1: Rolls[13] has associated the amygdala with the faculty for attaching value and thence with the approach or avoidance valence of a source of stimulus. Kling and Steklis[14] have reviewed the literature on the effects of ablation of the amygdala and attribute to this region, together with the orbital frontal cortex and temporal pole, the role of mediating affiliative behavior. Kling's studies report on the modulation of amygdalar discharges in freely moving animals living in social groups. The discharge rate increases during threatening experiences, and decreases during relaxed grooming which is an expression of attachment behavior.[15] Fight-flight behavior drives the sympathetic adrenal-medullary system. The perception of relaxation and peace, an accompaniment of the reverse process of grooming and attachment, probably leads to decreased sympathetic activity.

The arrow at the top of Figure 1 represents the response to perceived stimuli which work through the higher cortical mechanisms to initiate one or the other of the chains of response connected with the amygdala and the hippocampus. This diagram divides the mechanism of the stress response into two alternative channels: the one associated with threat to attachment and control, the other with actual perception of loss. When arousal is great and the stimulus is very strong, both mechanisms are simultaneously brought into play and differentiation is not feasible.[16] But evidence for the individual action of separate response patterns can be developed if appropriate experimental designs are used.

ANIMAL STUDIES DIFFERENTIATING STRESS RESPONSES

One clear demonstration of activation of the depressive pathway is in Sassenrath's[17] work showing how change of status affects ACTH. She studied the pairing off of a dominant rhesus male with one of several available female monkeys sharing the same cage. Sassenrath repeatedly measured the chosen female's 17-OHCS response to ACTH, finding that it decreased as she became increasingly secure in her relationship with the male. But another female not so favored showed a steadily increasing ACTH response during five weeks after pairing (Fig. 2).

Ely and Henry[18] found the same change in mice by placing males in complex population cages containing females. As males competed

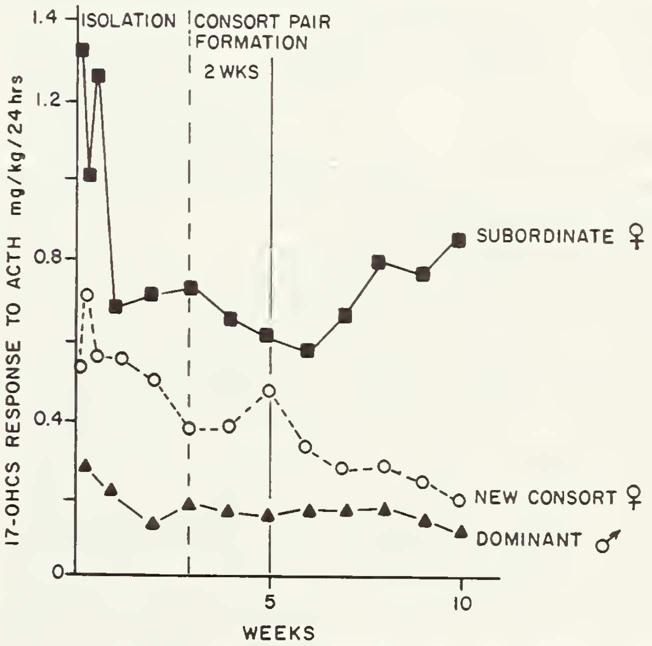

FIGURE 2. With the formation of a consort pair of rhesus monkeys, between three and five weeks, the new consort's 17-hydroxycorticosterone (17-OHCS) response to the adrenocorticotropic hormone (ACTH) fell, while that of a subordinate rival female rose as her status declined. The high status dominant male consistently showed a low corticosteroid response. (From J. P. Henry and P. M. Stephens: *Stress, Health, and the Social Environment*, Springer-Verlag, New York, 1977, Fig. 5-9, p. 82.) Reprinted with permission.

with one another during the ensuing weeks, a differentiation of plasma corticosterone was observed between the dominants and subordinates. The emerging dominant showed no significant change during the three-month observation, but the subordinates showed a statistically significant elevation of corticosterone. These males were not competing with sufficient intensity to induce significant changes in adrenal weight, but there was a difference in corticosterone level of those which were perceiving themselves as losing status and consequently were losing ready access to desiderata and control of the environment. A series of studies of dominant and subordinate males in mouse colonies established the reliability of this phenomenon (Fig. 3).

The adrenal medullary content of the enzymes tyrosine hydroxylase (TH) and phenylethanolamine N-methyltransferase (PNMT) was also measured in the same colonies of mice. Tyrosine hydroxylase, under sympathetic control, is the rate-limiting enzyme in the synthesis of noradrenaline and PNMT converts noradrenaline into adrenaline; thus, both are a measure of adrenal-medullary catecholamine synthetic activity. Furthermore, they correspond to active catecholamine levels. McCarty and Kopin[19] found that although rats with elevated levels of these two enzymes will have normal plasma catecholamines when at rest, they will have a greater increase in catecholamines than normal controls even from such a mild stimulus as being placed into another cage.

During experiments with the same mice, Ely and Henry[18] showed that TH rose significantly higher in dominants than in subordinates (Fig. 4). Meanwhile, PNMT not only increased in dominants but continued to rise, while falling in subordinates as they adapted to loss in status. Eventually mice in colonies adapted to each other, so these changes were not seen during the later stages of interaction. As the social hierarchy stabilized, social stimuli decreased in intensity and hormonal responses ceased. Thus, both adrenal-medullary activity and cortical activity became normal, suggesting that the emotional stimuli accompanying the animals' behavior had subsided. This was supported by behavioral evidence of lack of fighting (recent nips and bites on the tail and haunches).

The work just mentioned indicates that if the emotional state of an animal falls into a fight-flight stress response (corresponding to the left of Fig. 1), it differs biochemically from the depressive stress response. Although at least two forms of response to social-environmental threat can be differentiated, that is, a response to challenge and a response to perceived defeat, there will also be intermediate states combining both responses.

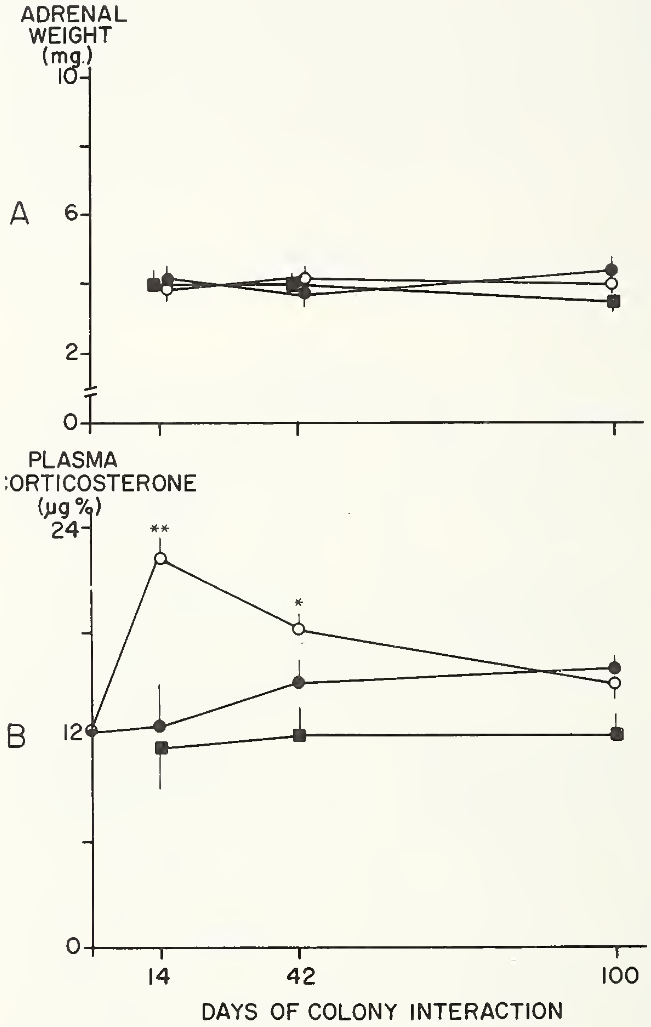

FIGURE 3. A contrast of adrenocortical responses of sibling males in a series of mouse colonies as they form a hierarchy of dominants and subordinates. Normal socialization was accelerated by having females in each colony. (A) The paired absolute adrenal weight of dominants (solid circles n = 4), subordinates (open circles n = 16), and controls (squares n = 6). Vertical lines are Standard Error of the Mean. The weights show no change, indicating that the social stimulus was not intense. (B) Plasma corticosterone of dominants (solid circles), subordinates (open circles), and controls (squares). The value for the still competing subordinates is significantly higher at 14 and 42 days. After colony dynamics stabilizes, this difference vanishes as the now thoroughly subordinated males accept the dominant. * = $p<0.05$, ** = $p<0.01$. (From D. L. Ely and J. P. Henry: Neuroendocrine response patterns in dominant and subordinate mice. *Horm Behav* 10: 156-169, 1978, Fig. 3, p. 162.) Reprinted with permission.

Figure 5 is a composite of data based on earlier studies of various laboratories; the data had been published without this demonstration in mind. The figure shows a gradual shift in the nature of the rat's response to 2-3 hours of immobilization each day, taped to a laboratory bench. After 40 daily exposures to immobilization, the rat's plasma corticosterone was only half as elevated as in the initial immobilization. Mikulaj and Mitro[21] comment that it was not caused by exhaustion of the hypophyseal adrenocortical system which still responded vigorously to a novel stimulus.

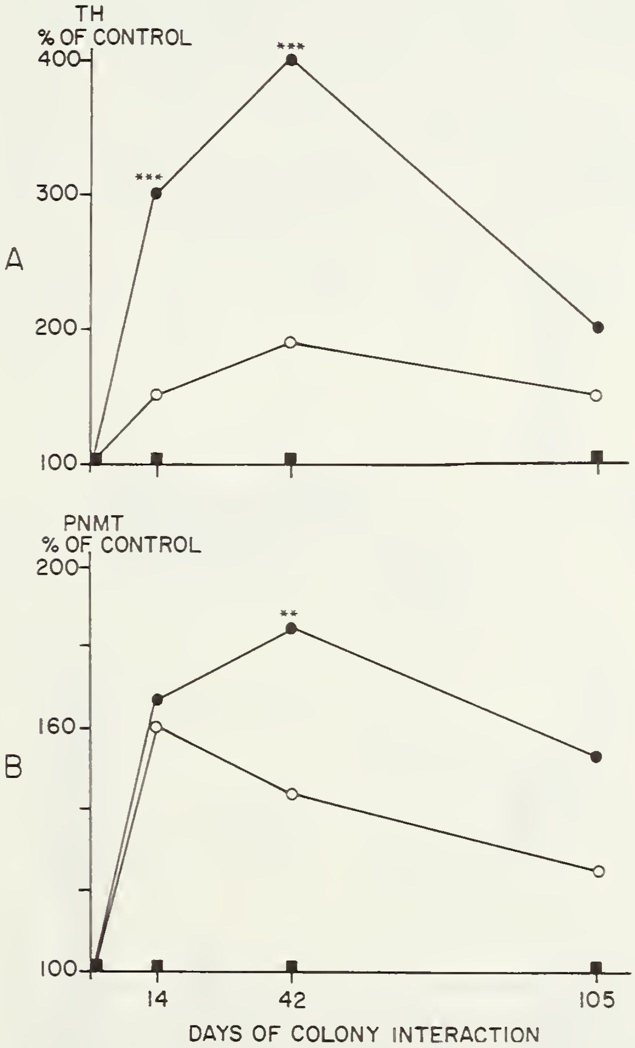

FIGURE 4. A further aspect of the study in Fig. 3 presenting the adrenal-medullary content of tyrosine hydroxylase (TH), the noradrenaline-synthesizing enzyme, and that of phenylethanolamine-N-methyltransferase (PNMT), the adrenaline synthesizing enzyme, in the adrenals of the same dominant and subordinate mice. (A) Percentage increase in TH in dominants (solid circles, n = 4), subordinates (open circles, n = 16), and controls (squares, n = 6). (B) Percentage increase in PNMT in dominants (solid circles), subordinates (open circles), and controls (squares). The data in both point to increased sympathetic activity in dominants. ** = $p<0.01$, *** = $p<0.001$. (From D. L. Ely and J. P. Henry: Neuroendocrine response patterns in dominant and subordinate mice. *Horm Behav* 10: 156-169, 1978, Fig. 2, p. 161.) Reprinted with permission.

Kvetnansky, Weise, and Kopin[21] showed that at the same time the corticosterone response was decreasing, adrenaline in the urine and noradrenaline synthetic enzymes in the adrenals were increasing with the daily immobilizations. A progressively enhanced ability to replace catecholamines was noted, despite a decrease in plasma cortiocosterone. Seemingly the rat changed his response to the experience. It can be argued that early immobilizations were regarded as a threat against which the rat was helpless and had no coping defenses. As both Weiss[22] and Seligman[23] point out, this perception of loss of control leads to the depressive response. Later, although just as noxious, the event is perceived as having a predictable outcome.

A study by Pfeifer and Davis[24] of the modification of the adrenal stress response by age and prior experience comes to similar conclusions. They showed that exposing mature male rats to repeated foot shocks failed to elevate tyrosine hydroxylase unless the subjects had been previously shocked. The prepared rat could better predict the outcome; if it responds with anger, the enzyme producing noradrenaline will increase. In keeping with the results just mentioned, the authors found that prior experience dramatically reduced the serum corticosterone response to shock, and by reducing the sense of helplessness, the stimulus to the adrenal-cortical system decreased.

The related studies of Corley, Mauck, and Shiel[25] reported on six groups of squirrel monkeys exposed to shock while confined to a pilloried chair. A monkey which could avoid shocks was yoked to one which could not avoid shocks they both received. Both were subjected to the Sidman avoidance test. The monkey which could avoid shocks developed nonlethal myocardial damage because they were exposed to sympathetic fight-flight activation, but more of the helpless monkeys died; the mechanism responsible, Corley and his associates concluded, was a parasympathetic response similar to that found in rats forced to swim for their lives. They argue that helplessness or inability to cope, characterizing the helpless yoked monkey, led to bradycardia that preceded death. Thus, they too have developed an experiment differentiating the sympathetic adrenal-medullary system from the adrenal-cortical response system.

HUMAN STUDIES DIFFERENTIATING STRESS RESPONSES

There is evidence that this differentiation of responses can be observed in humans. In the late 1950's, cardiologists Friedman and Rosenman came up with an effective experimental approach to the idea that psychological stress might be at work in coronary heart disease. They interviewed men and women, arbitrarily classifying them as having either a Type A or a Type B pattern of behavior: Type A's show an urgency of time pressure, competitive drive, and undue hostility when thwarted, but Type B's have none of these traits. Good predictive characteristics of Type A, mentioned by Schucker and Jacobs[26] in a recent study, are volume of voice and rapidity of speech and what they expressively call "plosiveness." Scherwitz, Berton, and Leventhal[27] also found that emphasis of voice and rapidity of speaking and answering differed quantitatively between Type A and Type B.

Persons with Type A behavior had twice the risk of coronary heart disease according to Friedman and Rosenman,[28] and biochemical differences in their responses to appropriate social stimulation were also found. Figure 6 that we have made up from data in reports published separately by Friedman and Rosenman's group shows: (A) when Type A persons receive a dose of ACTH, they excrete less cortisol than Type B persons[29] and (B) when both types are challenged by competition, the plasma noradrenaline of Type A rises from 0.41 to 0.54 mg/ml, but that of Type B remains unchanged[30] and in (C) the same response occurs during a normal working day — Type A excretes more noradrenaline in the urine, but Type B does not.[31] Thus the sympathetic catecholamine system of Type A responds more to the stimulus of competition, but the same stimulus may elicit a greater response of the pituitary-adrenocortical system in Type B.

Type A college students are more ambitious

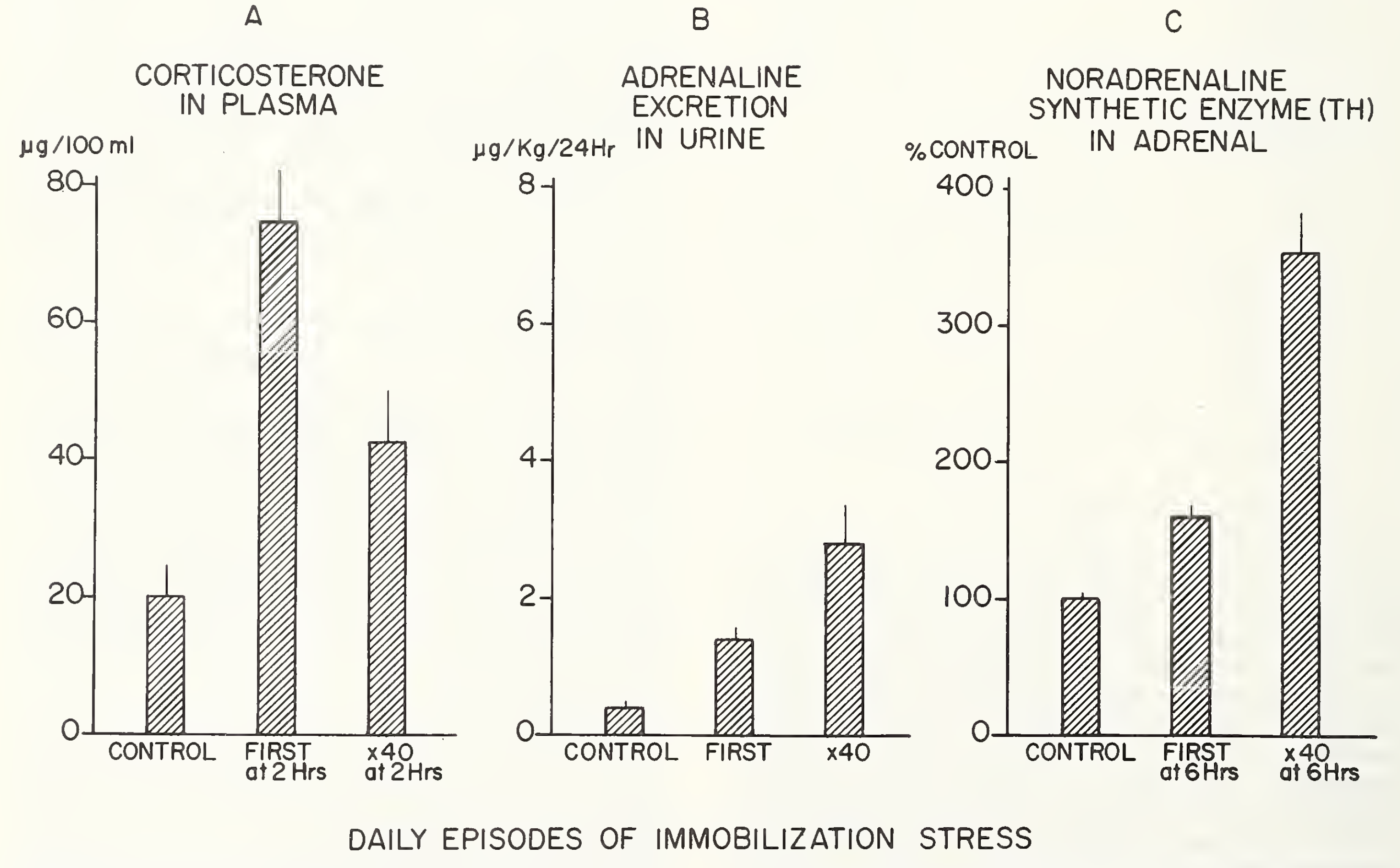

FIGURE 5. Composite diagram of data from three experiments by different research groups. In each, rats were exposed to approximately 40 daily episodes of immobilization stress, each lasting two hours. (A) Mean plasma corticosterone level after the fortieth episode was half of that following the initial immobilization. (B) By contrast, excretion of adrenaline in the urine was doubled at the fortieth episode ($p<0.001$). (C) Concentration of tyrosine hydroxylase (TH), the noradrenaline-synthesizing enzyme, in the adrenals was even more elevated ($p<0.001$). These changes indicate a shift from the stimulation of the adrenal-cortical to the adrenal-medullary system as the stimulus becomes more predictable. (From J. P. Henry and P. M. Stephens: *Stress, Health, and the Social Environment*, Springer-Verlag, New York, 1977, Fig. 7-9, p. 129.) Reprinted with permission.

according to Glass;[32] they work closer to the limits of endurance, earn more honors, and more go to graduate school. Moreover, they experience the loss of control as more threatening when defeated in spite of their best efforts. Glass observes that during their active coping efforts to achieve control, plasma noradrenaline may increase, but when they perceive control has been lost, they become passive and their noradrenaline may decline.[32]

Such changes are not limited to the sympathetic adrenal-medullary and the adrenal-cortical systems. There is evidence of an inverse relationship between gonadotropic hormones and the pituitary adrenal-cortical system's Selyean or alarm response. Christian[33] observed that gonadotropins decrease and ACTH increases among crowded, competing mice. Moreover, Bronson[34] has shown that the gonadotropic hormones of subordinate male mice decrease during fights to establish social order. The concurrent effect on androgens was the decreased size of the androgen-linked sexual preputial glands of the subordinates. Dominants show the opposite trend, having very large preputial glands.[34] It is relevant that both the men studied by Bourne[35] who were exposed to danger during combat in Vietnam but perceived high self-worth in the performance of military duty and the women awaiting biopsy reports of a lump in the breast studied by Katz and his associates[36] were shown to be protected by their defenses. These very different studies showed that feelings of social acceptance and high personal worth which commonly accompany effective psychiatric defenses are associated with decreased activation of the pituitary adrenal-cortical mechanism. Carroll's work[37] is compatible with this conclusion. He showed that primary depression accompanied by a lack of self-worth is associated with the opposite response of increased activation of the adrenal cortex.

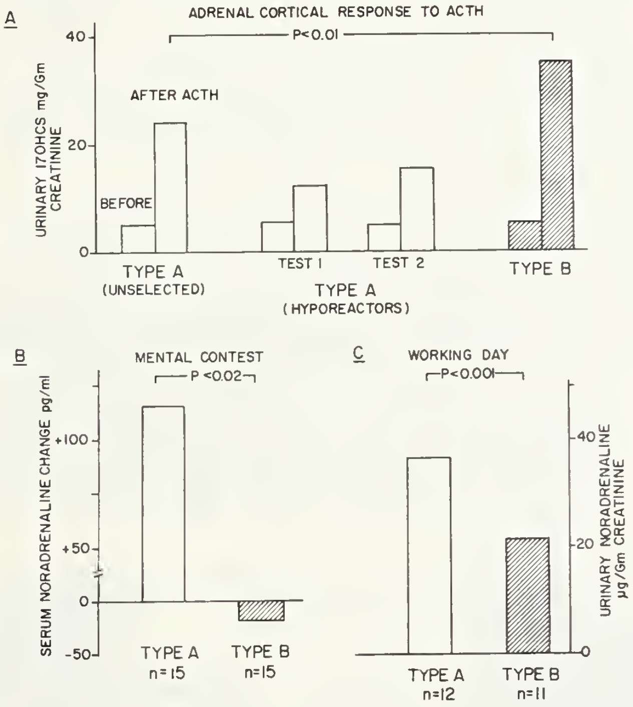

FIGURE 6. A composite of three experiments contrasting the neuroendocrine responses of subjects with personalities defined as Type A with those defined as Type B. (A) A fixed dose of the adrenocorticotropic hormone (ACTH) induced significantly more excretion of 17-hydroxycorticosterone (17-OHCS) in the urine of Type B's than of Type A's. Of 18 Type A persons, six were hyporeactors who hardly responded. (B) When Type A's were pitted against Type B's in a mental contest, their serum noradrenaline rose, while that of Type B's remained unchanged. (C) When the excretion of noradrenaline in the urine of Type A's was contrasted with that of Type B's during the working day, it was significantly higher. Taken together, these studies suggest that Type A's and Type B's differ in the responsivity of their sympathetic adrenal-medullary and pituitary-adrenal-cortical systems in the same way that dominants differ from subordinates. (From J. P. Henry and P. M. Stephens: *Stress, Health, and the Social Environment*, Springer-Verlag, New York, 1977, Fig. 7-14, p. 136.) Reprinted with permission.

An important study by Nuckolls, Cassel, and Kaplan[38] of women evaluated as having either weak or strong defenses during high or low life changes suggests that the human reproductive system is adversely affected by the depressing perception of high changes and weak defenses.

CONCLUDING COMMENTS

The physiological consequences of the arousal of the sympathetic adrenal-medullary system by the fight-flight response are not included because they were well described by Gilmore[39] in his paper on the cardiovascular response to emotional stress presented at the last conference. The frequent association of myocardial infarction with episodes, such as bereavement, may be a consequence of the depression response, depicted at the right in Figure 1. Glass's[32] observations that the Type A personality is vulnerable to depression are in accord with a study by Thomas, Ross, and Duszynski[40] of physicians with myocardial infarcts who readily became depressed even as students and likewise with a study by Bruhn and others[41] of the coronary prone as an effort-oriented person whose achievements give him little satisfaction.

It is not clear why myocardial damage should be more likely to occur during increased activation of the pituitary adrenal-cortical system and the mechanism connecting vascular damage with hypertension is not yet defined. We do know that immune response may be disturbed in these states and may induce auto antibodies against vascular tissue, and that the adrenal cortex, the thymus, and the reticuloendothelial system which are known to be intimately involved in the immune response are sensitive to the depressive type of stress.[42]

SUMMARY

(1) As a result of emotional arousal, purely cognitive processes can lead to the neuroendocrine changes of stress.

(2) Different psychosocial and physiological changes develop according to the emotion predominantly concerned. Anger and fear are associated with the fight-flight response, and the control animal's response to challenge results in sympathetic adrenal-medullary activation with the familiar chain of physiological changes.

(3) Depression is found in situations fraught with loss of control, as when an animal has recently been subordinated. By contrast, it involves activation of the pituitary adrenal-cortical system and depression of gonadotropins. ☐

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PATHOPHYSIOLOGY OF STRESS-INDUCED HYPERTENSION

ALVIN P. SHAPIRO, M.D.*

Hypertension is a multifactorial disease best defined as a disturbance in the complex regulatory mechanisms which control the relationship between the circulating fluid volume (i.e. cardiac output) and the space in which it circulates (i.e. peripheral resistance). The system includes renal, neural, hormonal, and structural mechanisms which in turn influence each other through multiple feedback devices to maintain normal blood pressure and oxygen delivery to peripheral sites in the body. Thus if "stress" is defined in the conventional engineering context as a "force which tends to deform a body," then all hypertensive disease could be considered as "stress induced."

However, the role of stress in hypertension has assumed a much narrower context, namely that noxious stimuli of behavioral origin have an impact on the initiation and perpetuation of this disorder and that their removal offers a therapeutic modality. The validity of this concept is supported by clinical experiences and by experimental studies in animals and man but the precise and quantitative nature of the impact of stress and the manner in which stress integrates with other stimuli in the production or maintenance of hypertension remains elusive. Reasons include problems in quantifying the effect of behavioral stimuli, the retrospective nature of most case analyses, and the difficulty in eliminating such stimuli or controlling them in an experimental context. Moreover, acute changes observed in blood pressure fail to establish whether chronic disease can be produced. Nevertheless, although stress, in common with most other causal mechanisms in hypertension, must rarely provide a single explanation for the disease, the available evidence has persistently suggested that the effects of behavioral change must be considered seriously among the variables which combine to explain the pathogenesis of this multifactorial disorder.

It should be understood that the primary mechanism involved in production of an effect on blood pressure by noxious stimuli of behavioral origin is neural and that the brain and central nervous system (CNS) play the role of the perceptive organs in exerting their impact. Yet it should also be clear that the CNS in turn mediates its effects through peripheral mechanisms which include renal, hormonal, and cardiovascular pathways. Similarly, the peripheral mechanisms in turn can influence the brain and behavior so that in a very literal sense the components of stress involved in hypertension are not unique, but parts of a finely tuned system whose disharmonies may evoke hypertensive disease.

The pathophysiology of stress-induced hypertension, therefore, must be examined in the same fashion that the pathophysiology of hypertension in general is approached. In fact, most all of the mechanisms involved in the control of blood pressure and the pathophysiology of hypertension which have been studied with respect to stress have been shown to be influenced by noxious stimuli. Such influences have been demonstrated in acute experiments and occasionally in chronicity studies.

Starting at the global level of the effect of the environment and the stresses arising therefrom on blood pressure, a number of epidemiologic studies affirm that hypertension is higher in populations which appear to be under exceptional stress. Thus blacks have higher levels of blood pressure in this country than whites. Studies in Detroit have demonstrated that socio-economic level among blacks inversely correlates with prevalence of hypertension. Elevated blood pressures have been associated with rapid cultural change in several areas of the world and with socio-economic mobility. The argument has been made that contemporary man is faced with the continuous stress of conflict and uncertainty which leads to cardiovascular adjustments appropriate to physical actions he might take in a primitive society but which he no longer can utilize in present day civilized society. Studies of chronic stress in animals ranging from noise and electric shock in rats, and avoidance conditioning in monkeys, to the elegant studies of social crowding in mice by Henry and his group have demonstrated the production of elevations of blood pressure. Common to many of these situations in both animals and man, the environmental stimulus usually involves the need of the organism to adjust to a stressful situation which has in it elements of constant threat and uncertainty, and the presence of an acquired or genetic cardiovascular or renal susceptibility.

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The brain, of course, is the perceptive organ for these environmental influences. The central nervous system and the autonomic nervous system transmit changes to the periphery which increase cardiac output and/or peripheral resistance. It is probable that the sympathetic nervous system and the release of norepinephrine are the key mechanisms involved. Much attention has been given to the peripheral mechanisms involved in norepinephrine release within recent years while increasing work has been done on the midbrain and medullary centers which mediate adrenergic discharge. The nucleus tractus solitarius in the medullary area of the brain is the relay station to stimuli coming from the periphery by way of the baro-receptors of the carotid sinus and passes these on to the vasomotor centers in the medulla. A variety of experiments have shown that the destruction of the nucleus solitarius leads to sustained lability of blood pressure and, in fact, in some animals to chronic hypertension.

Pituitary factors such as ACTH have long been known to induce peripheral changes in adrenal function which can affect blood pressure. Vasoconstrictor polypeptides such as vasopressin are released from the brain in response to stress and may well play a role in transmitting peripheral resistance changes. Similarly, other releasing factors from the hypothalmus may be indicted in such responses to stress although there are a paucity of such data.

One aspect of the brain's involvement in hypertension is the indirect effect of certain overt behaviors on blood pressure. Certainly the differences in salt intake in different populations can not simply be due to the availability of salt but are also due to salt-seeking behavior, a fundamental part of the development of the vertebrate from a marine animal to a terrestrial animal, and must play a role.

The pathophysiologic mechanism of hypertension which has undergone most intensive study in the past several decades is the renin-angiotensin-aldosterone system. Among the several factors which mediate renin release from the kidneys are autonomic nervous system stimulation and the presence of norepinephrine. Peripheral renin activity levels can be shown to be raised by stress in animals and in man; in turn, this will influence aldosterone and sodium metabolism. Furthermore, stress studies in rats have demonstrated increased vascular responsivity to infused angiotensin. Angiotensin also has effects on behavior in terms of producing thirst and apparently represents at least a primitive controlling mechanism over water intake. This is demonstrated clinically in patients with severe renin-dependent hypertension who suffer from inordinate thirst to the point where it is impossible to halt their seeking of water. This can result in marked overexpansion of the intravascular volume in such individuals. On the other hand, there appears to be an independent renin-angiotensin system in the CNS itself which could influence thirst and vasoconstrictor receptors, but which does not necessarily respond to peripheral stimulation. Recent work has suggested that peripheral angiotensin does not cross the blood-brain barrier. Still another angiotensin mechanism is suggested by the fact that large doses of angiotensin infused directly into the kidney actually cause natriuresis which is a further mechanism whereby the organism may adapt to stress. This may be due to elaboration of prostaglandin released under the influence of renin.

Many aspects of cardiovascular control, both central and peripheral, will respond directly to stress. Again usually these effects are through autonomic nervous system mechanisms. Stress will increase heart rate and also increase peripheral resistance by effects on beta-receptors and alpha-receptors respectively. Since blood pressure is the product of cardiac output and peripheral resistance, the relative proportion of each of these which is affected by stress and which vascular beds in the body are affected, determines the peripheral outcome as reflected in blood pressure. Of considerable interest are the data from a number of investigators indicating that cardiovascular adjustments may be exquisitely specific to different behaviors. For instance, studies by Zanchetti and co-workers have indicated that cats preparing to fight (agonistic behavior) display vasoconstriction and diminution of cardiac output; during defense behavior involving movement, they display increases in cardiac output and muscle blood flow. These specific patterns may arise from different centers in the brain and may interreact when the specific behavior involves several of the activities.

What ties all these phenomena together is the fact that most of the theories which have now evolved concerning the development of hypertension, whether it be essential (primary) or one of the secondary types, offer an integrated multifactorial etiology. Dr. Irvine Page, quite a few years ago, referred to this as the "Mosaic Theory." We have argued that there is a "plethora of causes" of hypertension; what we need is an understanding of how the various mechanisms integrate with each other. For instance, it is apparent that when various influences cause overexpansion of fluid and increased cardiac output there is a diminution in "space controlling" factors (vasoconstriction), as seen, for instance in falling levels of renin activity and norepinephrine. Similarly, when there are strong vasoconstrictive influences, volume and cardiac output may sharply decrease. Behavioral induction of hypertension probably represents situations in which stress has invoked massive activation of several of the systems which eventually cannot be counterbalanced, leading, perhaps, to renal functional impairment and its consequences or, as Folkow has described, to changes in peripheral resistance arising from increased flow through vessels so that a permanent state of increased vasoconstriction is established.

Thus, we need to look carefully at these field theories of causation of hypertension and further develop our understanding of where the system breaks down from unbalanced stimulation. This is now readily apparent in some types of hypertension. For instance, when an individual has a pheochromocytoma, the large amounts of norepinephrine which are released cannot be effectively counterbalanced. When an individual has a renal artery lesion releasing renin autonomously or a primary aldosterone tumor causing sodium and water retention, counterbalances eventually cannot resist these effects. Similarly then, the stress of severe turmoil, in a genetically endowed individual who has increased peripheral reactivity or excess salt intake, may lead to chronically elevated blood pressure. ☐

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PATHOPHYSIOLOGY OF CATECHOLAMINE-MEDIATED MYOCARDIAL DAMAGE*

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GALEN M. PIEPER, M.S.****
FRANKLIN C. CLAYTON, M.S.****

Information derived over the last decade indicates that myocardial necrosis can be characterized by any of several variant groups of morphologic features with different pathophysiological origins. The key to the understanding of these different forms of necrosis arises from the awareness of the stages preceding myocardial cellular death. This report concerns the manner in which stress influences certain types of cardiac death and to what extent it is preventable.

CORONARY HEART DISEASE

The temporal and spatial association between coronary thrombosis and myocardial infarction has suggested a direct cause and effect relationship. A number of discrepancies exist, however, with regard to the frequency, extent and distribution of coronary artery disease and myocardial infarction. Indeed, one can produce any percentage of incidence of coronary thrombosis in myocardial infarction by properly selecting the cases. For example, 86% to 96% of transmural myocardial infarctions will be associated with an acute thrombus.[1] Another example is the finding that more myocardial infarctions will be associated with an acute thrombus when patients survive 72 hours or longer after onset of symptoms of chest pain.[2] At the other extreme is a recent study in which 7% of patients dying of acute myocardial infarction had only minimal narrowing of coronary arteries.[3] Therefore, coronary occlusion, with or without thrombosis is not exclusively a precipitating factor, but may even be demonstrated to be a secondary phenomenon.

Another dilemma is the occurrence of numerous (37%) coroner's cases of accidental death without any prior history of cardiovascular disease but with documented obstruction of one or more coronary arteries.[4] Before their accidental deaths, one would have expected these cases to have had clinical manifestations of cardiac problems. Yet, such individuals appeared outwardly healthy. An example is that of an 83 year old woman who died in a pedestrian-vehicular accident. This person never had clinically defined coronary heart disease, yet postmortem pathology from plastic casts of coronary vessels revealed a completely obstructed coronary vessel.[5] Therefore, one may either live a completely "normal" life with obstructed coronary vessels or one may experience a myocardial infarction. Alternatively, it is possible to develop an infarction in the absence of any significant coronary artery obstruction. The factors which determine which fate predominates remain elusive. A lack of evidence for the precise role of obstruction, (thrombosis, embolization, spasm or hemorrhage into an atherosclerotic plaque), has required reevaluation of the validity of the traditional "Plumbers Concept" of myocardial ischemia and necrosis.

The dilemma may be somewhat resolved if one categorizes coroners' cases in relation to premortem symptomatology. Certain populations of patients dying from cardiac causes may be divided into at least two separate groups: those who die suddenly (seconds to less than 1 hour) and those who died less acutely (hours to days). Furthermore, a dramatic difference in pathologic features arises from selecting cases of those patients who die acutely (within 1 hour) with chest pain and those who died suddenly without prodromata. The predominant form of myocardial necrosis in the cases with antecedent chest pain is the classical category of coagulation necrosis which is characteristic of typical myocardial infarction. Unfortunately, the histopathologic features of this form of necrosis require 8 to 12 hours to develop.[6, 7] The hallmark is polymorphonuclear leukocytic infiltration. A major problem arises when trying to identify any pathological changes before 6-8 hours have elapsed. It is, therefore, not surprising that there is relatively little information available on the first 6-8 hours of myocardial necrosis in patients with acute myocardial infarction as compared to sudden coronary death.

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Some assistance in the identification of early coagulation necrosis has been provided by the study of Bouchardy and Majno.[8] They demonstrated that the presence of elongated, thinned wavy fibers and elongation of nuclei was an early manifestation of the loss of contractility and subsequent development of coagulation necrosis. The nonenzymatic histochemical technique of hematoxylin-basic fuchsin-picric acid (HBFP) stain has been reported to be capable of identifying myocardial ischemia as early as 30 minutes.[9] Unfortunately, at the light microscopic level, there are no other readily identifiable alterations apparent at the earlier time periods.

SUDDEN CARDIAC DEATH

In contrast, in a high percentage of cases of sudden cardiac death, the myocardium has a grossly different pathology. The normal striation pattern of sarcomeres is disrupted in focal areas of the myocardium. These disintegrated myofibrils form what has been frequently termed "contraction band lesions," which are the predominant feature of the coagulative myocytolysis form of necrosis. Furthermore, this form of necrosis was the predominant lesion in 76% of the cases of sudden cardiac death and was found to be the unique lesion in 67% of these cases of sudden cardiac death.[4] It is not known with certainty what effects these histopathologic lesions have on the production of sudden and lethal arrhythmias that are purported to occur in cases of sudden cardiac death, but the work of Lown et al.[10] has indicated a high incidence of ECG disturbances with many cases of sudden death.

This form of myofibrillar degeneration is characterized by coagulation of the contractile proteins from adjoining sarcomeres to form transverse contraction bands. This hyperfunctional form of necrosis has been described in clinical cases of pheochromocytoma,[11] experimental catecholamine-induced myocardial necrosis,[12] and various forms of stress.[13] As reviewed in the first National Conference on Emotional Stress and Heart Disease,[14] and summarized by Raab[15] "liberation of catecholamines, both from the adrenal medulla and from sympathetic nerve terminals, appears to be the common denominator of all stresses in the usual sense of the term."

Therefore, the lesions seen following stress, sudden cardiac death and/or catecholamine excess appear to be morphologically similar. This similarity led to an attempt to obtain a suitable experimental model to determine whether these stress- or catecholamine-induced lesions could be duplicated in the laboratory.

EXPERIMENTALLY-INDUCED NECROSIS

The administration of isoproterenol[12, 16] and norepinephrine to anesthetized, open-chested dogs has been used previously in this laboratory to produce these "contraction band lesions." These pathological features in dogs morphologically resemble the lesions of coagulative myocytolysis found in man.[17]

On a morphological basis, the isoproterenol infusions produced readily apparent lesions by one hour. Furthermore, recent evidence from this laboratory has indicated that these lesions develop very rapidly and can be readily detected within 5 minutes after initiating catecholamine administration. Significant numbers of these lesions were observed at infusion rates of 1.0 and 2.5 μg/kg/min. Similar lesions were seen in small numbers at the infusion rate of 0.1 μg/kg/min. They were seen rarely in hearts from control animals.

Detailed morphological and quantitative descriptions of these lesions are to be published shortly. Highlights of this study include the presence of two different types of lesions. The first and more obvious type consisted of a "total" myocardial cell disruption of contiguous sarcomeres apparently due to irreversible hypercontraction (Fig. 1). In addition to these massive lesions, there were smaller ones incorporating myofibrils of only a few sarcomeres adjacent to the intercalated disc. Thus, these latter ones have been termed "paradiscal" lesions (Fig. 2).

The paradiscal lesions were, in general, more numerous than the large lesions. Both types were distributed as individual cells or in small clusters of cells surrounded by "normal" tissue. While appearing to be randomly distributed throughout the left ventricle, both types were, in fact, more numerous in the subendocardial (inner) layer.

The nature of these structural anomalies was more clearly defined at the ultrastructural level. Myofibrillar disruption produced an electron dense region which, in the case of the "paradiscal" lesions, was a single band adjacent to and parallel to the intercalated disc (Fig. 3). Also, myofilaments in the remainder of this cell returned to a normal striation pattern within the space of a few sarcomeres. The "total" cell lesions exhibited clumping of myofilaments throughout the entire cell, forming a large number of individual transverse "contraction bands" (Fig. 4). Mitochondria in both forms of lesions were swollen and displaced from their normal position.

Concurrent with the pathogenesis of this condition was the expected hyperfunctional state produced by β-sympathomimetic stimulation.[16] Heart rate, in particular, rapidly increased upon isoproterenol administration (Fig. 5). Contractility (dP/dt) also increased (Fig. 6), but did not reach a maximum until after 15 minutes. Both of these indicators of mechanical overdrive re-

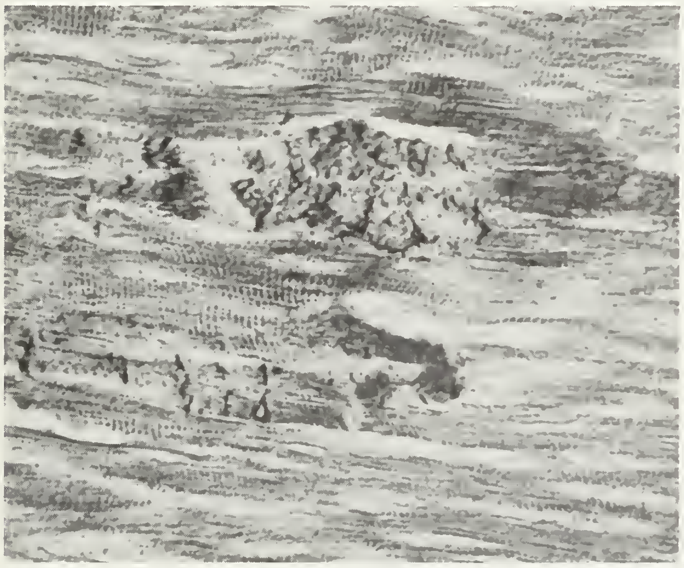

FIGURE 1. **Photomicrograph of small foci of "total cell" lesions involving coagulation of sarcomeres of entire cell to form numerous transverse bands. Foci of damaged cells surrounded by normal myocardium. PTAH X 208.**

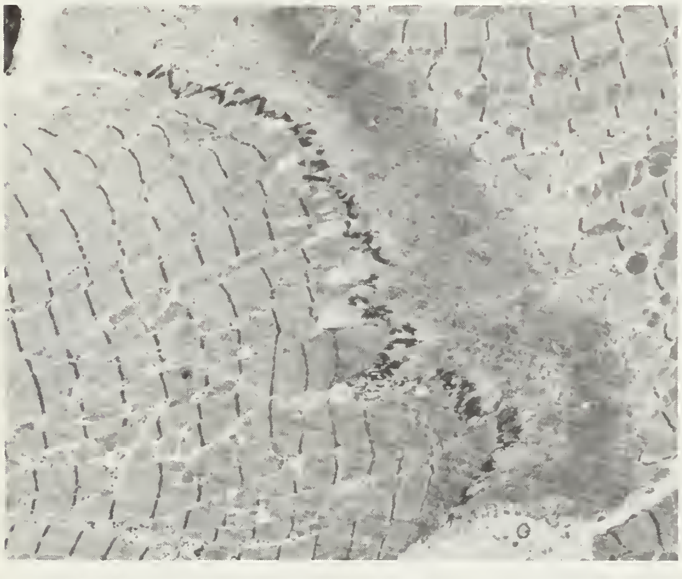

FIGURE 2. **Photomicrograph of cluster of "paradiscal" lesions involving coagulation of adjoining sarcomeres at the end of the cell adjacent to intercalated disc to form small homogenous transverse bands with a normal striation pattern throughout the remainder of the cell. PTAH x 208.**

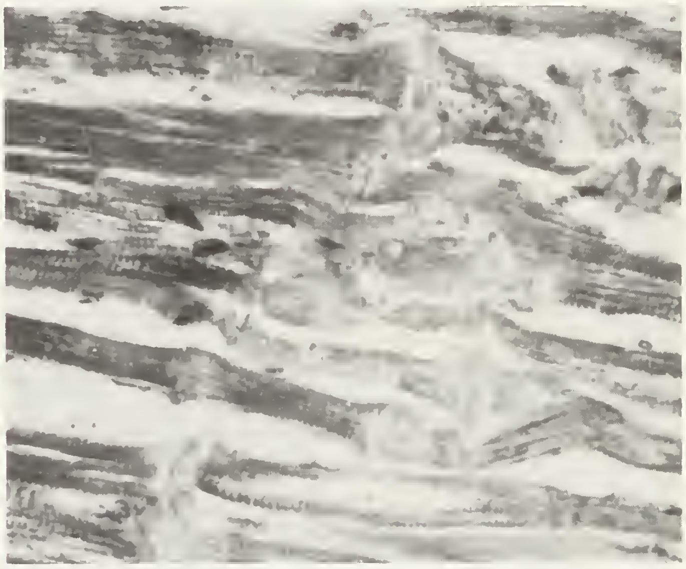

FIGURE 3. **Electron micrograph of small "paradiscal" lesion. Sarcomeres at the end of the cell coalesced into the transverse band adjacent to intercalated disc while sarcomeres in remainder of cell are normal. Uranyl acetate and lead citrate x 4200.**

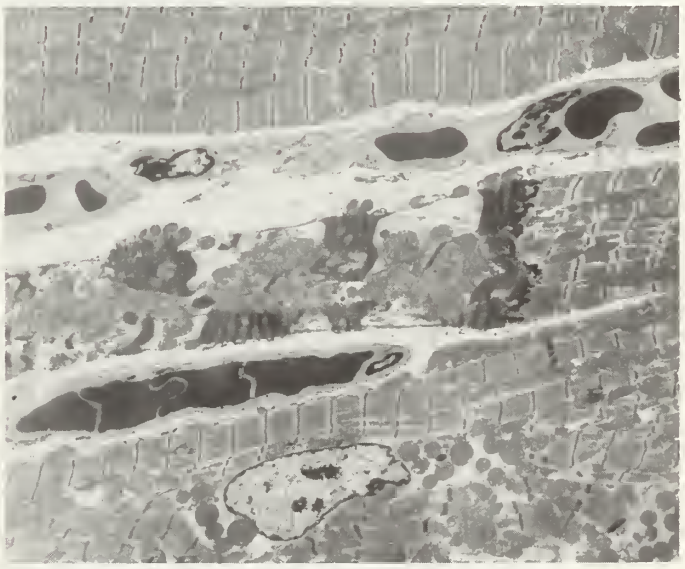

FIGURE 4. **Electron micrograph of "total cell" lesion with complete disruption of myofilaments to form the electron dense aggregations alternating with pale areas devoid of myofilaments. Uranyl acetate and lead citrate x 2940.**

sponded in a dose response fashion. Additionally, electrocardiographic aberrations were evident within the first minute of infusion and continued throughout the one hour of study.

Coronary blood flow, as measured by radioactively-labeled microspheres, increased upon catecholamine stimulation (Fig. 7). By 15 minutes, isoproterenol had increased blood flow to all three transmural layers of the myocardium, but there was a gradient in its transmural distribution such that the endocardial layer received relatively less blood flow as compared to the more superficial layers. These changes persisted throughout the remainder of the experimental period. Such a redistribution indicates "coronary steal" in the presence of increased flow.

Consequences of these hemodynamic variations were also evident on a biochemical level.[16] High-energy phosphates, the energy source of the cells, were greatly reduced by isoproterenol administration (Fig. 8). On a transmural basis, the greatest reductions occurred in the endocardial third of the myocardium. Concomitant with

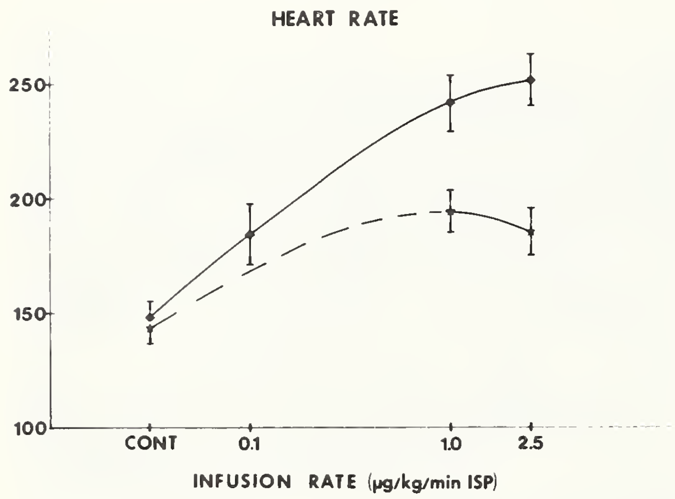

FIGURE 5. Differences in the maximum heart rates achieved with increasing infusion rates of isoproterenol (ISP) or isoproterenol preceded by a pretreatment with 0.4 mg/kg propranolol. Each point represents the mean of 5-6 dogs ± S.E. Propranolol was not studied with isoproterenol at a dose of 0.1 µg/kg/min. Isoproterenol alone represented by closed circles; Isoproterenol with propranolol represented by stars.

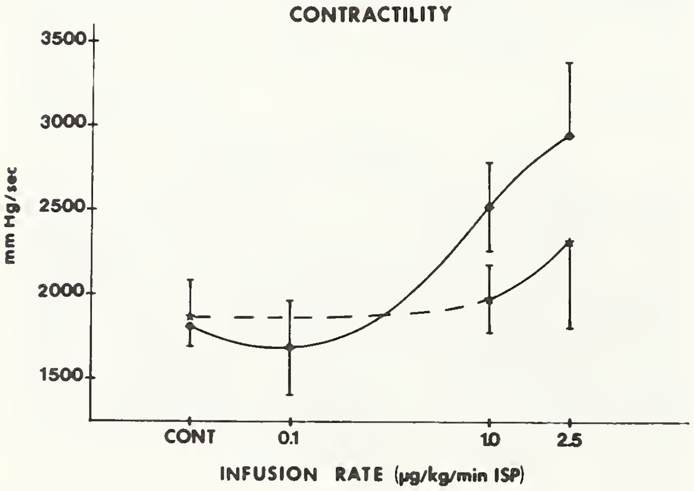

FIGURE 6. Increases in maximum myocardial contractility (dP/dt) with increasing infusion rates. See Fig. 5 for additional details.

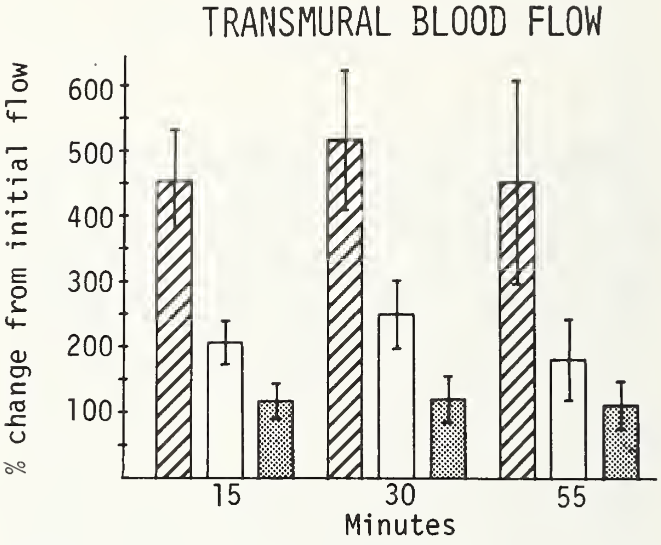

FIGURE 7. Change in the transmural distribution of left ventricular coronary blood flow following 2.5 µg/kg/min isoproterenol. Values represent the mean of 9 hearts ± S.E. Slashed bars = outer third; open bars = middle third; stippled bars = inner third.

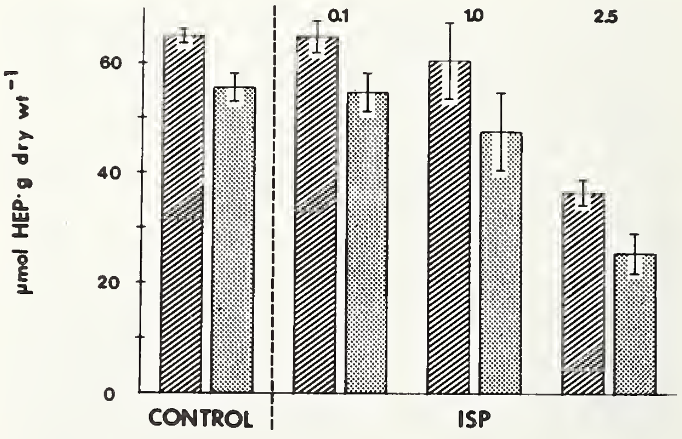

FIGURE 8. Cardiac tissue levels of high-energy phosphates (HEP = $\sum$ ATP and creatine phosphate) following various doses of isoproterenol (0.1, 1.0 and 2.5 µg/kg/min for 1 hour). Each bar graph represents the mean of 5-6 hearts ± S.E. Slashed bar indicates the outer third of left ventricle, while the stippled bar represents the inner third. Biopsy was taken from the posterolateral wall adjacent to the posterior papillary muscle.

this was a heterogeneous depletion of cardiac glycogen[16, 18] a major storage fuel in the heart. As with the high-energy phosphates, the inner layer was the most severely affected. Conversely, tissue lactate increased in response to isoproterenol with an extensive endocardial accumulation (Fig. 9). Thus, metabolically, the administration of isoproterenol produced a form of myocardial stress which had more pronounced consequences in the more vulnerable endocardial layer.

It then was necessary to determine whether a clinical dose of propranolol (0.4 mg/kg) could, in fact, block the detrimental effects of the higher, more pharmacological, dosages of isoproterenol. Interestingly, beta-blockade with propranolol at the lower of the two necrogenic doses of isoproterenol (1.0 μg/kg/min.) provided almost complete protection for most of the hemodynamic parameters (Fig. 5-6). The number of lesions appeared to be reduced, at this dose and the metabolic status of the heart remained at or near normal levels.[19] At the highest isoproterenol infusion rate, (2.5 μg/kg/min), propranolol pretreatment only afforded partial metabolic, morphologic and hemodynamic protection.[12]

These laboratory studies involving acute infusions of catecholamines have demonstrated the presence of readily identifiable lesions with both isoproterenol and norepinephrine which appear to be related to the dose administered, the redistribution in coronary blood flow and possibly to the hyperfunctional overdrive. There is also a disruption of normal metabolic states which along with the lesions, appears to predominate in the subendocardial third of the myocardium. As an initial step in identifying potential interventions, these preliminary studies with propranolol indicated that it may be of some benefit in alleviating the pathological consequences of this experimental condition.

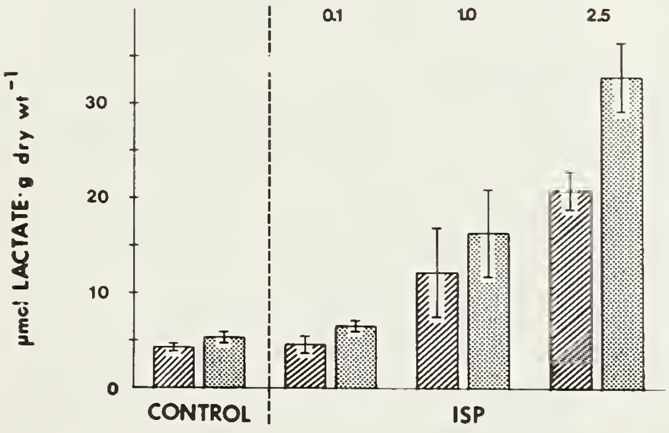

FIGURE 9. Concentration of myocardial lactate following various doses (0.1, 1.0 and 2.5 μg/kg/min for 1 hour) of isoproterenol. See Figure 8 for details.

In summary, lesions associated with sudden death in humans can be duplicated in animals employing both natural and synthetic catecholamines. Beta-blockade prevents their formation and maintains their numbers near control levels. As we begin to more fully understand the pathophysiologic sequence leading to the development of different forms of myocardial necrosis in the laboratory, there is a significant potential for the development of methods which may be useful in the clinical setting for the treatment and prevention of stress-induced cardiac necrosis and death. ☐

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The authors thank Mr. Jerry Lintz, Ms. Rosemary Nisi and Ms. Beth Leahy for their excellent technical assistance. The editorial assistance of Ms. Emily Salhany is also gratefully acknowledged.

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EPIDEMIOLOGY OF STRESS-RELATED CARDIOVASCULAR DISEASE

LEWIS H. KULLER, M.D.*

I. INTRODUCTION

The relationship between psycho-social factors and arteriosclerotic heart disease can be reviewed in several ways. First, the interaction between psycho-social variables and the pathogenesis of arteriosclerotic heart disease can be described (see Fig. 1 and 2). Second, the types of psycho-social factors can be measured and correlated with the incidence, prevalence and natural history of arteriosclerotic heart disease (Fig. 3).

There is overwhelming evidence that at least one psycho-social variable, so called Type A behavior as compared to Type B behavior is associated with a substantial increase in incidence, prevalence, mortality and probably extent of coronary atherosclerosis.[1] This relationship is consistent after adjusting for other known risk factors and has been partially substantiated at least by retrospective studies in other populations than the so called Western Collaborative Study Group. No other psycho-social variable has been consistently described as being a major risk factor for arteriosclerotic heart disease.[2] However, the failure to identify other consistent relationships may be a function of the type of study designs, poor measurement techniques and selection of the population at risk.

II. THE RELATIONSHIP BETWEEN PSYCHO-SOCIAL FACTORS AND THE NATURAL HISTORY OF CORONARY ARTERY DISEASE (Fig. 1)

A heart attack can be viewed as the culmination of a long incubation period disease "atherosclerosis," and a rapidly progressing phase leading to the heart attack.[3] The long incubation phase of atherosclerosis results in coronary artery stenosis. At the present time there are no good measurements of either the extent or the evolution over time of coronary atherosclerosis in an asymptomatic population. Most data describing the natural history of coronary atherosclerosis, therefore, depends on animal experimental studies, usually feeding of large doses of cholesterol or saturated fat, and more recently the measurement by coronary angiography of disease in symptomatic individuals. There have been several recent reports suggesting the association between Type A behavior and the extent of coronary artery stenosis at least as measured among individuals with symptomatic angina pectoris.[4] Although the studies are interesting, they do not answer the more fundamental question of the relationship between psycho-social factors and the progression of coronary atherosclerosis nor the relationship between any behavioral factors and the extent of coronary artery stenosis in asymptomatic individuals. Clearly, if there is a difference in the awareness of symptomatology, the likelihood of seeking medical care or coronary angiography may be related to behavior type. Perhaps then a relationship between behavior type and coronary artery stenosis among symptomatic individuals could be demonstrated.

The progression of coronary atherosclerosis to coronary stenosis to clinical heart attack cannot be measured over time in the same individual since this requires followup by repeat coronary angiograms in asymptomatic individuals. Thus at the present time animal experimental studies may be the only way of evaluating the relationship between various psycho-social factors and the progression of coronary atherosclerosis.

The precipitating factors for a heart attack have been evaluated both in terms of pathophysiology and precipitating environmental factors. Whether the same pathophysiological parameters are related to angina pectoris, myocardial infarction and sudden death is not known. Some investigators have suggested that instantaneous sudden deaths are not associated with any acute pathophysiological changes in the coronary arteries, while sudden deaths or acute myocardial infarctions may be more closely related to acute coronary thrombosis, hemorrhage or rupture of a coronary artery plaque.[5]

Recent clinical trials suggest that drugs that

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will prevent platelet aggregation and thrombosis may reduce sudden death among survivors of an initial myocardial infarction.[6]

Psycho-physiologists have correlated behavior with changes in the autonomic nervous system, levels of circulating catecholamines and thrombogenesis. There may be no acute pathology in the coronary arteries in sudden deaths following ventricular fibrillation (instantaneous deaths). An interesting hypothesis suggests that changes in the physical and social environment of susceptible individuals are the precipitant of cardiac arrhythmias, ventricular fibrillation and sudden deaths. The best example obviously would be the individual who was scared to death, and sudden deaths following severe emotional trauma.[7] Although such deaths have been described, the frequency in relationship to all sudden deaths occurring in the community is remarkably low.[8] Community studies of sudden deaths have generally been unable to identify a large number of cases in which a special psycho-social event was present. This remains an important question requiring further research.

Several investigators have attempted to objectively quantify specific life stresses prior to a heart attack.[9] These changes in life stress have been given specific weights and then life changes prior to the event are compared either between cases and controls or the recent as compared to remote events in the same individual.[10] Such studies depend both on the adequate recall of the events and the individual's perception of their significance. Also, a weighted system presuming that the events have equal importance for all individuals is clearly unlikely.

Another approach to the relationship between psycho-social factors and precipitation of heart attack has been to measure individuals during acute stressful events, either in the laboratory or in the community.[11] Early studies, for example, describe the cardiac arrhythmia during automobile driving in traffic[12] and during sexual intercourse.[13] More recent studies have attempted to recreate situations in the laboratory and monitor cardiac arrhythmias and other physiological changes. Some of these studies have further classified the subjects by behavior Type A and B or some other behavioral characteristic. It is obviously unethical to attempt in humans to create a true heart attack, ventricular fibrillation, or sudden deaths in the laboratory. The studies must presume that autonomic nervous system changes, premature ventricular contractions or other electrocardiographic abnormalities are harbingers of subsequent myocardial infarction and sudden deaths. However, whether these changes in the laboratory have any relationship to subsequent disease is unclear. These studies have also attempted to correlate changes in catecholamine levels with thrombogenesis, clotting, and possibly relate these to the precipitation of myocardial infarction and sudden death.

Most individuals who die suddenly or have a myocardial infarction have extensive coronary stenosis involving more than one coronary artery.[14] Any model that fails to consider the interaction of the extent of coronary artery disease with the possible precipitating events is probably irrelevant. Thus, laboratory attempts to create myocardial necrosis or ventricular fibrillation by severe social or physical environmental stresses among animal models with normal coronary arteries are clearly not testing the situation in man. Similarly, laboratory studies in young students testing the relationship between psycho-social factors and cardiac arrhythmias or changes in blood pressure or clotting may be of little relevance to the situation in man. Premature ventricular contractions, for example, are extremely common. There is no evidence at the present time that such premature ventricular contractions in normal individuals are harbingers of sudden death. On the other hand, premature ventricular contractions, especially those that are frequent and multi-focal, appear to be related to sudden death in individuals who have severe coronary artery disease, prior myocardial infarction, or angina pectoris.[15] Unfortunately the investigator in the laboratory is often limited to the study of "normal young healthy individuals" and may misinterpret normal physiological responses as predictors of subsequent disease.

III. STUDY DESIGN

The design of psychological and social factor studies in relationship to coronary artery disease can be divided into two broad categories (Fig. 3): studies of personality and behavior and studies dealing with social and environmental factors. The personality-behavioral factor study can generally be subdivided into four broad categories (Fig. 4): 1) those studies which have attempted to correlate standard psychological testing instruments such as the Minnesota Multi-Phasic Personality Inventory, etc., or other standard measurements and the risk of heart attack or natural history of disease[16] and 2) studies which have developed a structured questionnaire of use specifically for cardiovascular research. The best examples of such instruments are the Jenkins Activity Scale which was developed from the behavior Type A-B personality interview[17] and a modification of the Framingham Type A-B questionnaire.[18] Another example might be the questionnaire related to anger and hostility developed for the studies of blood pressure in Detroit by Harburg and others.[19] The third type of study uses basically an unstructured direct interview approach; the classical example is the Type A-B interview format developed by Drs. Friedman and Rosenman. Although the questions themselves are structured, it is primarily the responses and the answers to these types of questions which determine the classification of Type A or Type B. The questionnaire was also developed in response to observations in cardiac patients rather than as a part of a standard psychological measuring instrument as used in other studies.[20] The fourth type of study is a mixed group which depends on both a structured standard psychological measuring instrument and an instrument developed specifically for cardiovascular disease. The best example of this is the variety of techniques used in the Framingham Study to evaluate psycho-social factors.

The social environmental type studies can also be divided into five broad categories (Fig. 3). The simplist types of studies were those relating social class as defined by occupation,[21] income, education, etc., with mortality from coronary artery disease.[22] Substudies have now also tried to relate social class to the incidence and natural history of disease. The second group of studies related a specific occupational stress to the risk of disease.[23] The third attempted to measure the effects of change in the environment on the population at risk. These studies have usually concentrated on the changes from a rural to urban environment or on rapid changes within an urban environment.[24] The studies were initially started by the late Dr. John Cassel and involved primarily populations in rural North Carolina undergoing substantial urbanization. The fourth type of studies are similar to the environmental studies but in this case instead of the environment changing, the individual moved from a traditional to a non-traditional society. Studies of the populations in the South Pacific migrating to New Zealand and Australia are classical examples of this type of study.[25] The studies of Japanese migrants to Hawaii and California may also be considered as part of this type of study.[26] The hypothesis is that the social and psychological changes in individuals migrating from a traditional society to a non-traditional society will result in increases in stress and risk of heart attacks. The fifth type of social environmental studies relate to the concept of social status incongruity.[27] The incongruity may be either one or two generations, that is, an incongruity between an individual's job and education or between husbands' and wives' educations or jobs. Two generational incongruity may result from upward social mobility such as a comparison between the blue collar background of a father and a white collar occupation of a son or daughter. The initial studies were done primarily in North Dakota and California by Syme and others.[27]

A. *Behavior – Personality*

Several years ago Dr. Carolyn Thomas of the Johns Hopkins University attempted to develop a model to explain the possible relationship between psychological factors and high blood pressure.[28] We have attempted to expand on this model to look at the relationship between behavior, risk factors and disease (Fig. 5).

The first hypothesis suggested is that certain risk factors such as high blood pressure, cigarette smoking or elevated levels of serum cholesterol lead to atherosclerosis and heart attack and that following the heart attack there are certain behavioral changes which are then measured and correlated with the disease (Fig. 6). This model presumes that the disease itself, that is the clinical heart attack, is the cause for the change in behavior. This hypothesis does not seem tenable at least for the one major behavioral characteristic, Type A and Type B. The long term prospective studies such as the Western Collaborative studies have clearly demonstrated that the behavior pattern existed prior to the onset of clinical disease. However, many other studies that attempt to measure behavior among individuals who have had a heart attack were probably biased because they measured the effects of the disease on the behavior and rather than behavior as a causal factor in the disease. It would seem that most behavioral studies of coronary artery disease will have to be prospective or experimental to test the hypothesis relating behavioral factor to disease.

The second hypothesis suggests that the risk factor results in both a change in behavior and a change in the risk of disease. In this case, the risk factor leads to behavioral change and disease at approximately the same time (Fig. 7). Again, the data from the Western Collaborative Study would tend to negate this hypothesis, at least in relationship to Type A-Type B behavior. However, studies of patients in coronary care units or shortly after a myocardial infarction to determine the various psycho-social factors related to the precipitation of a heart attack are likely to fall into the trap and presume that the behavior is related to etiology of disease rather than the fact that disease and behavior are approximately related to certain risk factors which may have lead to the disease itself.

The third hypothesis suggests that genetic characteristics may result in a certain type of behavior and also in the progress of risk factors which lead to disease (Fig. 8). The presumption here is that the behavior may precede the disease and be identified as a risk factor in prospective studies and yet basically have nothing to do with the etiology of the disease. For example, there is a distinct possiblity that certain genetic determinants of the host response to the environment may have a major effect on behavior and, at the same time, these genetic determinants of the host response may lead to changes in neurohumoral factors that could precipitate a heart attack. Thus in this model the neuro-humoral factor (i.e., such things as elevated catecholamines, or increased autonomic nervous system activity) was the risk factor for a heart attack and may be correlated with behavior, but the behavior has little or nothing to do with the risk of a heart attack. No studies have yet proven or disproven this hypothesis. Probably the only way that this hypothesis can be tested is by experimental manipulation of the behavior, the subsequent determination of changes in various neuro-humoral factors, risk factors and disease. Such a study would be extremely difficult at the present time, because there is little evidence that behavior can be modified in a large number of individuals. Also, the measurement of the neuro-humoral factor is difficult and the significance of the biochemical parameters in relation to actual neuro-humoral activity is not certain. Obviously, such a study would have to be open ended and could not be double blinded. The effects of change in other risk factors would be a major confounding variable. It would, therefore, seem probable that descriptive genetic studies and studies of behavior in childhood would be the next step in studying this model.

The fourth hypothesis suggests that a risk factor may lead to a certain behavior and that these same risk factors may be related to disease perhaps many years later (Fig. 9). Prospective studies would suggest that the behavior occurred before the disease and that individuals with the specific behavior would have an increased risk of disease, but as is the case in the third model, the behavior would not be causally related to disease and modification of the behavior would have little to do with reducing the disease. Could some undetected risk factor such as a deficiency or excess of an important trace element or micronutrient result in certain behavioral changes and also increase the risk of heart attack? The solution to this problem is not simple. We could continue to search for the "origins of the specific types of behavior" and attempt to determine in animals and perhaps in man the relationship between various environmental factors and behavior, or again turn to clinical trials to determine the effects of the behavioral change on risk of disease. At first, this hypothesis would appear to be rather far fetched. However, the effects of lithium in affective disorders does raise the possibility that either excess or deficiency of various trace elements or micronutrients could have a profound effect on behavior and conceivably on the risk of disease.

Hypotheses five and six are quite similar (Fig. 10). Model five suggests that certain aspects of our behavior interact with the environment and have a profound effect on risk factors and that this effect on the risk factors resulted in subsequent disease. Hypothesis six suggests that behavior directly relates to the risk of disease independent of a step through the known risk factors. With regard at least to the behavior Type A and B situation, this hypothesis, appears to be the most probable at the present time. The test of this hypothesis initially requires replication of the relationship of specific behavior type and disease in other populations using longitudinal prospective study designs. In spite of the frequent reports that the Type A-B hypothesis has been replicated in other populations, the Western Collaborative Study remains the only long-term prospective evaluation of this hypothesis. Most of the other studies are either cross-sectional or case control type and are subject to the biases previously described. The second and far more important approach will be clinical trials to determine the effects of changing the behavior on both the risk factors and also on subsequent disease. An alternative will be those studies currently being done in the Multiple Risk Factor Intervention Trial[29] in which the risk factors such as blood pressure, smoking, and cholesterol are being modified among individuals who have both Type A and Type B behavior measured by either the Rosenman-Freidman Questionnaire or the Jenkins Activity Scale. One might hypothesize that the behavior is independent of the known risk factors and that individuals with Type A behavior who have their other risk factors modified, will not have a reduction of risk of heart attack while individuals with Type B behavior who have a similar reduction in other risk factors will have a substantial reduction in risk of disease. However, even the most zealous believers of the Type A-Type B relationship probably do not believe that the A-B behavior and other psycho-social factors are completely independent of other known risk factors and that modification of major risk factors would have no effect on the risk of disease.

One of the most intriguing questions concerns the relationship between behavior and the known risk factors (Fig. 11). Most of the studies of behavior have noted little or no relationship between the levels of the risk factors and behavior except at extreme levels of behavior A or B where there might be a small relationship to the levels of the risk factors. The interaction between personality and environment is obviously the major determinant of the level of the risk factors. Why do some people smoke and others not smoke? Individual food preferences determine the types of nutrients that people consume and may relate to the cholesterol level in the blood. Some individuals exercise and others do not. Yet most studies fail to show a significant relationship between behavior, at least behavior Type A and B, and the level of risk factors measured at one time in a so-called clinic situation.[30] Some studies suggest that behavioral change, independent of other environmental modifications such as dietary patterns, resulted in a change in level of risk factors such as a rise in serum cholesterol at the time of income tax season, independent of a change in any nutrients in the diet.

Short term changes in a risk factor such as the daily up and down of serum cholesterol level or even weekly variations may be related to the behavior of the individual either directly, as suggested by the accountant studies,[31] or because of modifications of the individual behavior in response to an environmental stress. Thus, we could hypothesize that in a certain environmental situation or stress an individual with a specific type of behavior changes his diet. This dietary change may result in a short term gain in weight, positive caloric balance, increased consumption of saturated fat and cholesterol, and a subsequent increase in the serum cholesterol level. Thus, when individuals have to work long hours, are threatened with time restraints, or major job responsibilities, they may consume pastries, candy, increased amounts of coffee, alcoholic beverages, and change their level of lipoprotein and other biochemical parameters. When this adverse environmental situation ends, they may return to their normal diet patterns and, within a short period of time, the serum cholesterol and triglycerides may return to the previous levels. Failure to measure the individual during the environmental stress situation or shortly thereafter may result in the presumption that the level of the risk factor and the behavior are not related. The repeated changes in the risk factors in response to the environment in an individual with a specific type of behavior may be the major factor which enhances the progression of atherosclerosis and even a heart attack. Thus, although statistically the behavior type appears to be independent of the known risk factor, it is still very possible that the interaction between the behavior, the social environmental situation and the risk factors may be a major determinant of the risk of heart attack. Studies which will measure the levels of the risk factors at frequent intervals in the same individual and determine both the behavior type and other psychological correlates and the environmental situation including careful measurement of nutritional intake, smoking behavior, exercise, weight change are necessary prerequisites to the test of this hypothesis. Until these studies have been done and have clearly shown whether there is a relationship between behavior and risk factor change over a short term and if such changes are then related to the risk of heart attack, the presumption that the behavior type is independent of the risk factor type is a statistically proven hypothesis but not a biologically acceptable one.

B. *Social Environmental Studies*

The social environmental studies can be broken down into a variety of categories as previously mentioned in Figure 3. Unlike the studies of the behavior type and risk of disease, many of the social environmental studies lack precision in study design and generally are either cross-sectional or retrospective studies and are based usually on mortality analysis rather than on incidence of disease.

1. Social Class:

The inverse relationship between social class and coronary disease has been recognized for many years. The reason for this inverse relationship between social class and coronary artery disease has never been fully explained. At least three factors might be involved: differential level of risk factors; exposure to environmental precipitants of heart attack; and utilization, availability and quality of health service.

Several recent publications have suggested that individuals classified as being in the lower social class have a higher frequency of risk factors especially cigarette smoking, blood pressure and even serum cholesterol levels. The relationship between social class, environmental pollutants, and risk of heart attack has never been effectively studied. Obviously, poorer people tend to live in areas where levels of air pollution, crowding, and poor environmental conditions are prevalent. Whether the high levels of these air pollutants may be associated with increased mortality from coronary artery disease has not been substantiated. Our previous studies in Baltimore suggested no relationship between levels of carbon monoxide in the ambient environment and subsequent mortality from coronary artery disease. Other studies, however, in Los Angeles have suggested such that a relationship may exist.[32] It is also distinctly possible that certain occupations may be associated with an increased risk of heart attack. Unfortunately, studies of environmental exposures, secondary to occupational environment as a risk factor for heart disease are few, and if such a relationship exists, it certainly has not been identified at least in relationship to any large number of workers.

A recent study from the Health Insurance Plan (HIP) of Greater New York suggests that individuals in the lower social class have a poorer prognosis after a heart attack.[22] They noted, for example, that individuals who had complex premature ventricular contractions, on a one hour tape recording taken several months after a heart attack had an excess mortality. However, the excess was primarily limited to those individuals in the lower socio-economic class. Since one might presume that within the HIP group the quality of medical care was quite similar for all social classes, the reasons for these differences are unknown and unexpected. However, differential use of medical services within the pre-paid medical care plan could conceivably account for the differences. Habits may differ between the two groups; for example, a higher proportion of individuals in the upper social class may have quit smoking or have other behavioral changes which are effective in reducing their risk of subsequent mortality following a heart attack.

Further investigation of both the environmental and social factors that distinguish people in higher and lower income groups would clearly be worthwhile. Special attention is probably needed in relationship to environmental precipitants of a heart attack and also to the interrelationship between habits, utilization of health services and subsequent morbidity and mortality from heart disease.

2. Occupational Stress:

The second type of social and environmental studies were those which attempted to demonstrate a specific type of occupational stress in relationship to heart attack. A classical example of such studies is the work of Henry Russek[23] and others which attempted to describe certain occupations considered to be stressful and associated with higher prevalence of heart disease. Many questions remain unanswered, as it is extremely difficult to separate the physical and the psycho-social environmental stresses. There is also the problem of job selection and the perception of occupational stress by the host. Some individuals working in a certain type of environment which may appear to be stressful to the outsider may find this to be a very rewarding and relaxing type of occupational activity. Other individuals may find that their occupation, although not stressful, may be so unappealing as to create a serious occupational stress. There is also the problem of job mobility. Individuals who are healthy and may have low risk factors may be upwardly mobile while individuals who develop heart disease or have certain unusual behavioral characteristics may be less mobile.[33] Comparison of workers within various occupational groups may therefore result in spurious conclusions.

Perhaps the most important area that needs further study is the relationship between a specific occupational stress or occupational situation and the possible precipitation of heart attack, especially sudden death. Many examples could be considered. For example, there is very little data on the effects of shift work on the risk of a heart attack. Do individuals who move from one eight hour shift to another at frequent intervals have an increased risk of heart attack because of possible changes in their diurnal rhythm of hormones or other factors? Do individuals who are forced to work overtime at frequent intervals have an increased risk of heart attack during these periods of increased work load? Do individuals who are exposed to jobs which are basically hazardous have an increased risk of heart attack? One interesting recent example is the relationship between noise and cardiovascular diseases. Several studies have suggested that individuals exposed to noise have increased levels of elevated blood pressure.[34] One study has suggested a correlation between hearing loss and level of blood pressure among selective service registrants.

The fundamental question in many of these occupational studies relating noise to either blood pressure or to heart attack is whether the physical exposure to noise is the risk factor or whether the stress related to continual exposure to noise is the risk factor. Furthermore, individuals exposed to noise in the environment may also be at risk because of the particular type of work that creates the noise. Thus the noise may be just a secondary variable in relationship to other job related stresses.

3. Rural to Urban Environments:

The observation that as an area became more urbanized the rate of coronary disease within the population increased was championed by the late Dr. John Cassel from his studies in North Carolina.[24] Relatively few investigators in the United States have pursued this line of investigation. The continued rapid change in the Southeastern United States and perhaps in the Southwest may offer the potential for further study. Changes from rural to urban environments in other countries may be a better model for this type of study. The reasons for increased risk of disease with urbanization have not been fully explained. Several hypotheses are prevalent. Increasing urbanization results in a certain amount of stress being placed on individuals from rural upbringing and that social stress plus the increased competition for jobs, crowding and the general upward mobility of the population all cause an increased risk of heart attack. A second possibility is that the effects of environmental change on the individual's behavior may convert individuals who might be considered rural Type B's to urban Type A's. Furthermore, with urbanization there may be an independent change in the level of the risk factors related to dietary modification, elevations of serum cholesterol, increased prevalence of cigarette smoking, perhaps more obesity, higher salt intake, and higher levels of blood pressure. The levels of physical activity may also decrease in association with changes in jobs, lack of availability of recreational facilities and reduction in the amount of free time to participate in various types of recreational activities.

Finally, one of the problems in doing these types of studies is to properly characterize the population. With urbanization, there is immigration and sometimes it is difficult to determine whether the increase in disease rates is due to the migration of a higher risk population into the area. If further studies are to be done to look at the question of environmental change, several factors must be taken into account. First, the populations must be identified prior to the changes from the rural to the urban environment. Once the event has occurred, it is very unlikely that any useful information can be collected. Ideally, it is necessary to have a comparison group, that is a group which is rural and in which no changes occur and an urban population in which no changes occur. It would be ideal to study a population which moved from an urban to a rural setting, but in the United States that appears to be extremely unlikely. Once the populations are identified, it is important to study the changes in risk factors, behavior and activity before and after the change, and then compare the differences between those that have stayed rural, those who were initially urban and stayed urban, and those that changed from rural to urban. Over time, it may be possible to measure the incidence of heart disease, the case fatality, and subsequent death rates within these populations.

The relationship of disease within these populations could then be related to the level of risk factors. Short of this approach, it appears very unlikely that the interrelationship between urbanization and risk of heart disease will be adequately resolved.

4. Migrants:

Individuals migrating from a so-called traditional to a non-traditional society have been noted to have an increase in heart attacks. Most of these so-called traditional societies have very low rates of heart attack, so generally we are comparing individuals who migrate from low incidence to high incidence areas. It has also been suggested that those who migrate and maintain their traditional roots have a lower risk of disease than those individuals who adapt to the non-traditional culture. Furthermore, the studies of the Japanese Americans in San Francisco suggest that even after adjustment for the usual risk factors, the risk of disease may be higher in those who have changed, but have not acculturated.[35] There are several problems in these types of studies. The presumption that those who have maintained their traditional lifestyle have a lower risk of heart disease independent of usual risk factors would appear to be dubious. Clearly, those individuals who have maintained their traditional life style have different risk factors and different habits than those who have acculturated more rapidly. Individuals who have maintained a more traditional life style are more likely to have a slower acceptance of new habits, change in diet, smoking and other factors. The current levels of the risk factors in those who have maintained traditional and non-traditional existence may be quite similar but those who have maintained their traditional style have probably changed more recently and therefore have a shorter exposure time (incubation period). Long term studies are necessary, including studies of children and careful observation of the changes in the risk factors over time in those who have maintained the traditional and non-traditional life styles. Several studies have suggested that individuals who attend church may have a lower risk of heart attack. It might be useful to determine whether individuals who maintain orthodox behavior irrespective of their particular religion may also have a lower risk of heart disease. Certainly the risks are lower among Mormons and Seventh Day Adventists, but this is generally believed to be related to their habits rather than to the religious philosophy and social behavior within these religions. It would be interesting to study Orthodox Jews, very religious Roman Catholics and perhaps some of the Orthodox Eastern religions and determine whether these groups have lower rates of disease as compared to the more liberal members of the religious sect.

5. Social Status Incongruity:

Is there an increased risk of heart attack associated with social mobility? If a husband and wife are from markedly different social origins, is the husband more likely to have a heart attack? Do men who come from rural backgrounds and have urban white collar jobs increase their risk of heart attack as compared to men who have an urban white collar background?

Several investigators have suggested that individuals who have so called social status incongruity either generational, that is between parent and offspring or cross-sectional, between their education and current occupation, have an increased risk of heart attack.[36] It might be presumed that such incongruity is associated with some aspect of stress and that this results in an increased risk of heart attack.

Unfortunately, none of these studies have attempted to define the stress which may be related to social status incongruity. Rather, these studies have measured the situation and have not defined the possible pathophysiological mechanism for the increased risk of heart attack. These studies also have not looked at the specific behavioral characteristics of the individual. It would obviously be very important to try and correlate social status incongruity with other measures of behavior. One might presume, for example, that individuals who are Type A may have a greater upward social mobility and, perhaps, greater cultural changes and increased social status incongruity. Some studies have attempted to relate social status incongruity to the classical risk factors and also to the subsequent risk of heart attack, but the sample sizes have been very small. The ongoing studies may provide larger sample sizes and a more heterogeneous population to test this hypothesis further.

Further studies of this problem might include the determination of social status incongruity by various interview techniques along with subsequent measurement of behavioral characteristics and risk factors and hopefully the ultimate measurement of risk of disease. An alternate approach might be to determine the frequency of various types of social status incongruity among certain types of social behavioral characteristics, i.e., behavior Type A and B. Finally, it would be important to try and determine what factors in the environment, in relationship to social status incongruity, might be associated with changes in either risk factors, behavior type, or risk of disease.

IV. SUMMARY

The current interest in psycho-social factors and coronary artery disease can be attributed to several factors. The establishment of a relationship between Type A and B and risk of disease prospectively has clearly moved the methodology from the pseudo-scientific to the scientific, testable arena. The possibility that there may be identifiable precipitants for heart attack and the improved ability to study neuro-humoral and electrophysiological parameters has opened up new areas of research. Finally, the need to better define the psycho-social parameters in order to intervene in other risk factors such as diet, smoking and exercise has been recognized.

The next steps are to improve methodology; replicate the current findings, especially with regard to Type A and B behavior in other populations; and to try and link the psycho-social and physiological parameters. Finally, much greater emphasis should be given to the measurement of the psycho-social factors in their natural environment, especially the interrelationship between behavior, risk factors and pathophysiology in the environment of the individual. Neither the measurement of the relationship between psycho-social factors and other risk factors in the clinical situation or the measurement of behavior and pathophysiology among young normals has the potential for improving our understanding of disease pathogenesis. ☐

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FIGURE 1

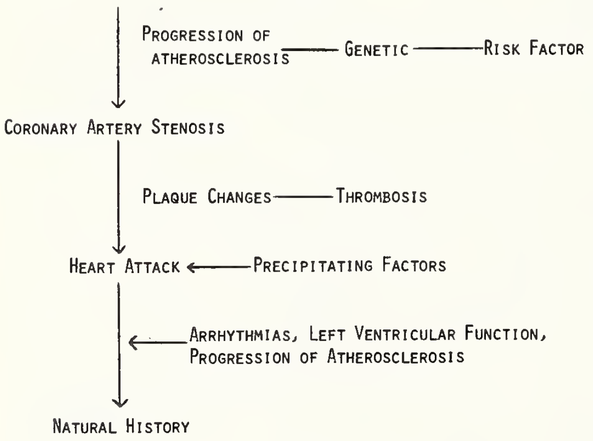


FIGURE 2

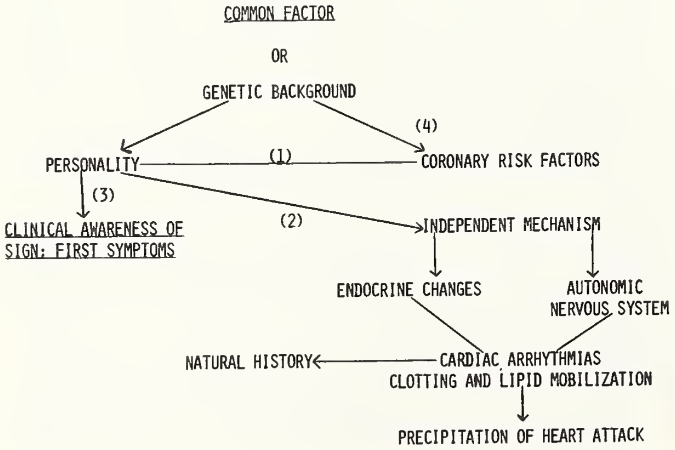


FIGURE 3

DESIGN OF PSYCHOLOGICAL AND SOCIAL FACTORS STUDIES

PERSONALITY - BEHAVIOR	SOCIAL ENVIRONMENTAL
1. STRUCTURED STUDIES USING STANDARD PSYCHOLOGICAL MEASURING INSTRUMENT	1. SOCIAL CLASS
2. STRUCTURED STUDIES USING INSTRUMENT DESIGNED FOR CARDIOVASCULAR RESEARCH	2. OCCUPATIONAL STRESS
3. UNSTRUCTURED - DIRECT INTERVIEW	3. ENVIRONMENTAL CHANGE
4. MIXED GROUP	4. MIGRANT
	5. SOCIAL INCONGRUITY

FIGURE 4

DESIGN OF PSYCHO-SOCIAL RISK FACTOR STUDIES

A. PERSONALITY - BEHAVIOR:
  1. STRUCTURED STUDY
    A. STANDARD PERSONALITY TYPE QUESTIONNAIRE, (M.M.P.I.)
    B. STRUCTURED FOR CHD: JENKINS ACTIVITY SCALE
  2. RELATIVELY UNSTRUCTURED
    TYPE A-B
  3. MIXED TYPES
    A. STRUCTURED PERSONALITY AND CHD ONLY - FRAMINGHAM

FIGURE 5

POSSIBLE RELATIONSHIP BETWEEN PSYCHO-SOCIAL FACTORS AND DISEASE

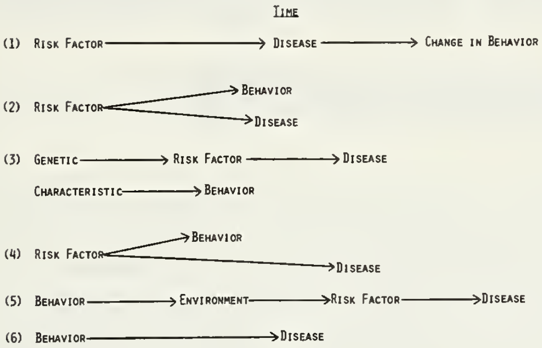

Time
(1) Risk Factor → Disease → Change in Behavior
(2) Risk Factor → Behavior → Disease
(3) Genetic → Risk Factor → Disease
Characteristic → Behavior
(4) Risk Factor → Behavior → Disease
(5) Behavior → Environment → Risk Factor → Disease
(6) Behavior → Disease

FIGURE 6

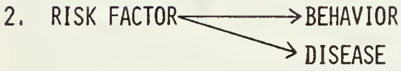
POSSIBLE RELATIONSHIP BETWEEN PSYCHO-SOCIAL FACTORS AND DISEASE

1. RISK FACTOR → DISEASE → CHANGE IN BEHAVIOR

   A. Not tenable at least with regard to clinical disease: Long term prospective study such as Western Collaborative clearly demonstrate that behavior pattern present prior to clinical disease:

   B. Many other studies comparing heart disease patients, behavior and controls probably biased:

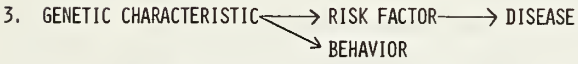
FIGURE 7



A. Results of Western Collaborative Study would tend to abrogate this hypothesis:

B. Interview studies of patients in coronary care units or shortly post-MI are probably biased:

FIGURE 8



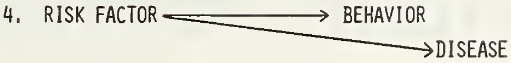
A. Disturbing possibility that genetic determinants of response to environment have major effects on behavior and some environmental factors could lead to heart attack:

   1. Need to Study: Familial distribution of behavior: (Genetic studies)

   2. Development of "Behavioral patterns in childhood":

   3. Relationship between behavior and genetic markers:

FIGURE 9



Could "some" undetected risk factor, i.e., deficiency or excess of important trace element or other micronutrients result in change in behavior and also increase risk of heart attack? Prospective studies would identify the risk factor prior to disease:

Either further search for origins of behavior or clinical trials to determine effects of behavior change on risk of disease will test this hypothesis:

FIGURE 10

5. BEHAVIOR → ENVIRONMENT → RISK FACTOR → DISEASE

6. BEHAVIOR → DISEASE

Most tenable hypotheses at present: Behavior such as behavior type A or B directly related to risk of heart attack:

Test of hypothesis requires; (1) Replication in other populations using longitudinal or prospective study designs; (2) Clinical trials to determine effects of change in risk factors on subsequent disease:

FIGURE 11

POSSIBLE RELATIONSHIP BETWEEN BEHAVIOR AND KNOWN RISK FACTORS

1. Interaction between personality and environment is major determinant of risk factors, i.e.,

   Why do some people smoke and others not smoke?
   Food Preferences:
   Exercise:

2. Most studies fail to show a significant relationship between behavior using behavior type A-B etc., and levels of risk factors; measured at one time in a "static" situation:

3. It has been suggested that behavior change independent of other environmental agents can change the level of a risk factor; i.e., serum cholesterol:

4. Short term changes in a risk factor may be related to behavior directly or by modification of habits:

Relationship between behavior and known risk factors:

A. Stress → Dietary Change → Increase in Serum Cholesterol

B. Stress → Increased Cigarette Smoking → Increased Levels of Carboxyhemoglobin, etc.

5. The repeat changes in the risk factors may enhance the progression of atherosclerosis and heart attack:

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EVIDENCE FOR THE RELATION OF PSYCHOSOCIAL FACTORS TO CORONARY HEART DISEASE

C. DAVID JENKINS, Ph.D.*

Emotional stress and heart disease — is the relation between these conditions a new idea or a very old one? Really it is both. Recent issues of medical journals are documenting in a quantitative way the contribution of behavioral factors to cardiovascular diseases, particularly coronary heart disease (CHD). At this conference you have already heard about many of the newest developments in this exciting area of research.

The basic idea of an association between emotions and cardiovascular function goes back historically much further than most people realize. Three hundred fifty years ago a young physician with new and wild ideas wrote the following: "Every affection of the mind that is attended with either pain or pleasure, hope or fear, is the cause of an agitation whose influence extends to the heart." The year was 1628; the Latin monograph in which this appeared was *Exercitatio De Motu Cordis et Sanguinis in Animalibus*, and the author's name was Dr. William Harvey.

While William Harvey's observation applied to the general functioning of the heart, a famous British surgeon 150 years later saw the connection between his own experience with angina pectoris and his emotions and behavior. Dr. John Hunter (1729-93) once stated, "My life is in the hands of any rascal who chooses to annoy me. . . ." Dr. Hunter died suddenly while attending a board meeting at St. George's Hospital in London in 1793.

A hundred years later, another of the immortals of medicine, Sir William Osler, was among those to observe that the then quite rare condition of atherosclerosis and its attending episodes of angina pectoris were not spread randomly throughout the community, but rather tended to attack a specific type of person. Osler wrote in 1897 ". . . I believe that the high pressure at which men live, and the habit of working the machine to its maximum capacity are responsible for (arterial degeneration) rather than excesses in eating and drinking. . . ." Other early examples could be cited supporting the basic hypothesis that emotions and behavior relate to the presence of coronary disease.

In our century, Osler's description of the coronary patient striving for success in a responsible but high-pressured way was elaborated upon by the famous American psychiatrists, the Menningers, who saw in coronary patients strong agressive tendencies, which were often strictly repressed. Kemple affirmed this pattern of aggressiveness in his observations of coronary disease patients and added that they were usually very ambitious and strove compulsively to achieve goals incorporating power and prestige.

As one reviews the broad array of published articles dealing with psychological and social variables and their relation to atherosclerosis and coronary disease, a very clear pattern emerges. Those psychosocial factors which most closely and most intensely involve sustained overstimulation of the central nervous system have the most consistently positive associations with risk of coronary heart disease. Those psychosocial factors which only at some times or only in some persons imply chronic agitations of the central nervous system have weaker or less consistent relationships to increased coronary risk. And finally, those variables most removed from central nervous system involvement have the weakest associations.

To illustrate this more specifically, one finds that the strongest, most consistent correlates of CHD risk across a variety of research circumstances are the following: disturbing emotions, such as anxiety and depression; interference with sleep; perhaps an exaggerated blood pressure response to the cold-pressor test; and finally — with the greatest amount of research support — the Type A coronary-prone behavior pattern.

Having somewhat less strength and consistency as a predictor of CHD risk are such variables as work overload, life dissatisfactions and chronic conflict situations. Finally, such social structural variables as social mobility, migration, educational level and number of life change events are less certain to be uniformly a cause for emotional arousal or distress and concomitantly are less consistently and less strongly associated with risk of coronary disease in recent studies. It may be that the socioenvironmental factors raise CHD risk only in some persons and under circumstances where they result in prolonged conflicted arousal or distressing emotions. Thus, except for behaviors that influence standard risk factors and those sociocultural variables which influence environmental exposures, it appears that all the remaining psychosocial risk factors operate through the central nervous system to generate changes in autonomic nervous functioning and endocrine activity which in turn have a cumulative impact upon the cardiovascular system.

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I. RESEARCH EVIDENCE THAT ANXIETY, DEPRESSION AND SLEEP DISTURBANCE ARE CHD RISK FACTORS.

Most research evidence for the relation of emotional distress to CHD comes from retrospective studies. A great many of these studies have found that patients with coronary disease have elevated levels of anxiety. Fewer studies have utilized measures of depression or general neuroticism but these reports reveal the same trends.

When one considers only prospective studies, the amount of evidence narrows sharply. I am aware of less than ten such reports. The general thrust of these studies is that anxiety, depression and general neuroticism are prospectively associated with the future development of angina pectoris and possibly also with coronary death, the latter reported from one large study of 8,000 twin pairs from Sweden. Neuroticism, anxiety and depression however, are not predictive of future myocardial infarction in any of the large scale studies where these factors have been measured.

I have recently discovered, however, that a variety of studies have inquired about sleep disturbance. Some have asked about insomnia, others about restless sleep or fatigue upon awakening. Despite the differences in methods, six such studies have consistently shown that problems in sleeping are a precursor of coronary disease. In most research reports this finding is buried amongst a large number of variables and only recently has this consistency become clear.

Mayou describes sleeplessness as particularly characteristic of patients with angina, and Thiel found it more common in patients with myocardial infarction than in controls. Bengtsson and colleagues asked their female subjects whether they had experienced stress, anxiety or sleep disturbances related to interpersonal conflicts. Women with CHD were much more likely than healthy subjects to have reported such problems continuously for a year or more.

Three prospective studies have made similar observations. Floderus noted that difficulties "in falling asleep" and "often being restless" were specific items associated with later development of angina pectoris and total mortality (myocardial infarction could not be determined because follow-up ECGs could not be obtained). Gary Friedman found that persons in the Kaiser Permanente population who reported trouble falling asleep or staying asleep were more likely to have myocardial infarction later. Similary, Thomas and Greenstreet observed that medical students who reported being tired upon awakening were heavily represented among the small number who had incurred myocardial infarction at the time of a follow-up study. The variable of disturbed sleep should receive greater attention by physicians doing risk factor determinations for coronary heart disease.

A number of research reports have appeared implicating the stress associated with life change as a risk factor in coronary disease. Many of these reports have used lists of life change items and observed greater numbers of reported life changes in the most recent six months or one year as compared to earlier periods in the CHD patient's lives. It should be noted that most of these studies do not involve a control group, but rather utilize patients as their own controls. All their data are gathered at one point in time however, and these studies have not taken adequate account of the substantial amount of forgetting which takes place when persons are asked for recollections of events in more distant time periods. Two separate studies, one by Uhlenhuth and colleagues and the other by Jenkins and colleagues, have shown a "rate of forgetting" of about 5% of life change units per month for at least the first six to nine months which have elapsed after events have occurred.

The two large life change studies with the strongest research design have failed to find scores from life changes schedules to be predictive of CHD. Lundberg et al. used infarction patients and a matched control group in a retrospective study. They found no statistical differences between the two groups when total life change units were calculated by the standard method, but they found significant differences when each person assigned to the events experienced the scores which reflected his own perception of the impact of those events. In the largest prospective study of the life change hypothesis which has been published to date, Theorell and colleagues have followed 6,579 Swedish construction workers for twelve to fifteen months. They found no association between elevated life change scores and incidence of acute myocardial infarction over that time. Elevated scores, however, did predict development of neuroses and certain other chronic disabling conditions.

II. THE TYPE A CORONARY-PRONE BEHAVIOR PATTERN: DEFINITION AND EVIDENCE FOR ITS VALIDITY.

Of all the postulated behavioral risk factors for CHD the coronary-prone behavior pattern, Type A, has thus far amassed the greatest amount of research evidence. This evidence fulfills many of the demands which the scientific method requires of a relationship which is judged "casual" in nature.

Dr. Meyer Friedman defines the coronary-prone behavior pattern — also called Type A behavior — as "A characteristic, action-emotion complex which is exhibited by those individuals who are engaged in a relatively *chronic struggle* to obtain an *unlimited* number of *poorly-defined* things from their environment in the *shortest* period of time, and, if necessary, against the opposing effort of other things or persons in this same environment." In his recent lectures, Dr. Friedman asserts that the most critical aspects of Type A behavior are excesses of aggression, hurry, and competitiveness, all of which are acted out as a struggle to overcome barriers in the environment.

People who manifest this behavior pattern are called Type A individuals, and those who have the opposite type of behavior, a relaxed, unhurried, mellow, satisfied style, are designated Type B individuals. The Type B is also interested in progress and achievement in most instances, but tends to flow with the stream of life more of the time, rather than constantly struggling against it as the Type A does.

A supplementary definition of the behavior pattern is as follows:

The coronary-prone behavior pattern is considered to be a style of living characterized by extremes of competitiveness, striving for achievement, aggressiveness (sometimes stringently repressed), haste, impatience, restlessness, hyperalertness, explosiveness of speech, tenseness of facial muscles, and feelings of being under the pressure of time and under the challenge of responsibility. Persons having this pattern are often so deeply committed to their vocation or profession that other aspects of their lives are relatively neglected. Not all aspects of this pattern need be present for a person to be classified as Type A. This pattern is neither a personality trait nor a standard reaction to a challenging situation, but rather it is the reaction of a psychologically predisposed person to a situation which challenges him. Different kinds of situations evoke maximal reactions from different persons.

It is important to distinguish Type A behavior from the concept of stress. The term "stress" is used in a variety of ways, sometimes to refer to a painful stimulus or upsetting situation, and at other times to refer to a personal reaction or alarm, discomfort, or pain. In contrast, the Type A behavior pattern is neither a stressor situation nor a distressed response. It is rather a style of overt behavior with which some people confront life situations, either pleasant or troubling, provided that some element of challenge is felt to be present.

Detection of the Type A pattern is based *only* on overt behaviors. It does not depend on inferences about underlying traits or psychodynamics. The latter become important, however, when we deal with the issue of changing the behavior pattern.

To open the discussion, let me explain briefly the measurement of the Type A behavior pattern. There are two primary ways in which Type A has been measured repeatedly in past years. There are additional ways of measuring this pattern which have been used only once or twice, and more ways are being developed. The first major method is the structured interview procedure developed by Dr. Meyer Friedman and Dr. Ray Rosenman. This interview is not just a collection of data, but is a special interaction between two people, a study subject and a trained interviewer who covers certain areas of inquiry, and also covers it in a specific style. It is a challenge interview. It is a miniature experiment in provoking Type A response from a respondent. There are well established training programs for persons to learn the techniques for administering the interview and interpreting it. Rating of a person depends as much or more on the style with which he or she responds to the interviewer than it does on the actual content of the responses.

The second procedure for measuring Type A behavior was developed, not to replace the interview, but to be used in large-scale studies. This technique is a self-administered, multiple-choice questionnaire which is then scored by computer. Scores are calculated and statistically adjusted to a common measurement metric and these are printed. These are numerical scores on continuous scales, similar to numerical reports of blood pressure or cholesterol. The questionnaire technique is called the Activity Survey. The scoring norms were developed from the Western Collaborative Group Study population. For ease in interpretation of scores, the neutral point or mean of the population was set at zero and the standard deviation at ten for all scales. Positive scores signify the Type A direction, negative scores the Type B direction, and that is the metric used in the tables reporting Activity Survey results.

TABLE 1.
Identification of Type A and Type B Subjects by the JAS Type A Scale in a Cross-Validation Sample of 419 Men

Type A Score 1965 JAS	Classification by 1960 and 1962 WCGS Interviews		Percentage rated in agreement
	Type A*	Type B*	
+10.0 and over	75	7	91
+ 5.0 to +9.9	50	11	82
0.0 to +4.9	31	24	56
− 4.9 to −0.1	43	33	43
− 5.0 to −9.9	19	41	68
−10.0 and less	10	75	88
Overall percentage in agreement	68	78	73

* Number of individuals.

We've talked about the two techniques for measuring Type A behavior, the structured interview of Rosenman and Friedman, and the Activity Survey or JAS as it is labeled in these tables. The first question is, do these two procedures measure the same thing? Table 1 shows the results of a test of how we categorized people in a cross-validation sample. This group was not utilized in the development of the scoring system. In this sample, the 1965 edition of the JAS, Type A scale was used to score people who were classified by the interview rating they received both in 1960 and 1962.

People do change over time and some of the lack of agreement here is due to changes over time. We tried to minimize this by making sure the people were consistent over at least two points in time by the interview, then we looked to see how the Activity Survey score came out three years after the second interview. The overall agreement is 73% between the two techniques. The JAS did very well in predicting the extremes, about 90% in each instance. However, the JAS did not agree well with the interview when scores were divided at the median. Agreement would have been higher if we could have set the cut-point between Type A and Type B at a minus 5 JAS score rather than zero, but the division of the scale at zero was an *a priori* decision and we adhered to it.

Now, we know that the JAS agrees with the interview but the question remains: Does this computer scored questionnaire correlate with anything with regard to heart disease?

By the time of the 1965 examinations of the WCGS, some of the men had already developed coronary disease since the beginning of the study. Those who survived and remained in the study in 1965 took the test along with everybody else. We learned about a year later that they

TABLE 2.
Mean JAS Scores of Male Survivors of CHD for 104 yrs. Compared to Random Sample Free of CHD, Western Collaborative Group Study

JAS Scale	CHD Cases N=98	Free of CHD N=511	t-test	P*
Type A	2.58	−0.46	−.270	.005
Speed & Impatience	1.13	0.28	−.075	.250
Job-Involvement	−1.83	0.08	1.71	.050
Hard-Driving	3.31	−0.17	−3.17	.005

* One-tailed probabilities

were included. We sorted them out and compared them with a control group free of CHD and found that these CHD cases are a little over a quarter of a standard deviation above the mean and the healthy controls are slightly below the mean. This represents a statistically significant Type A score differential between these two groups (see Table 2). The groups are not significantly different on the speed and impatience factor, and for job involvement, the difference is in the "wrong direction." Men with coronary disease are less job involved. That may or may not be the result of their illness. The CHD cases are, however, significantly more hard-driving, competitive and conscientious. These significances are beyond the .005 level. Of course, this relationship may be somehow secondary to coronary disease also. It's unlikely, however, that someone who is ill will be saying, for example, that they are hurrying more or they're working longer hours or they're more concerned about precision, or that they're more impatient. It's possible but you'd expect those kinds of questions to be answered in the opposite direction if someone was slowed down by illness. But let's keep that possible source of bias in mind.

So far we've spoken mainly about the Western Collaborative Group Study conducted in California. And so, we ask the question, can this finding be replicated in some other place? Our next study was conducted across the continent in Bridgeport, Connecticut. The study groups were men and women who were all hospitalized, either for coronary disease or for some other noncardiovascular condition. Again, this is a retrospective study but it is in a different place and it was conducted by a different team of investigators. I was not directly involved in this study except to provide the investigators with tests. Dr. Zyzanski scored them blindly and did the data analysis after the cardiologist in Bridgeport, Dr. Licciardello, identified the CHD cases. The results are shown in Table 3. These hospitalized cases scored a quarter of a standard deviation above the mean of the normative population. The controls are much more Type B. These CHD cases are very high on hard-driving conscientious characteristics, and the controls are significantly lower. The Type A relationship is at the P = .02 level. The Hard-Driving factor differs at P = .001. Ages are different also, with the CHD cases being older. We performed a two-way analysis of variance, stratifying by sex and controlling for age, and the same results came out as strongly as before. The question as to whether people become more Type A simply because they're sick, or whether somehow the fact of illness may influence these scores, is answered to some degree by having hospitalized controls rather than healthy controls. Now we see that in fact generally these hospitalized people are lower on three JAS scales than the mean of the healthy sample from California. Of course, we are dealing here with a different population, socioeconomically and geographically.

It would be useful to replicate these findings in still another geographic area, in a different institutional setting and with different investigators. This next study came from a VA hospital in Florida. Doctor Charles Spielberger was involved in the study with Doctor David Hiland, who actually collected the data and performed the analysis. It was found once again that the Type A score is significantly higher in CHD cases than in the control group composed of men hospitalized at the same VA Hospital for noncardiovascular illnesses. The speed and impatience factor (S) is extremely low in the control group and moderate in the CHD cases. The hard-driving factor (H) is not significantly different between groups, but the CHD cases are higher than the control group. The CHD patients are higher both on state anxiety and trait anxiety. Both these differences are significant, supporting our earlier references to observations that anxiety tends to be higher in coronary disease patients, particularly after myocardial infarction. These studies are replications supporting the validity of the Type A relationship to CHD, at least within the United States. But, will this replicate once we change cultures or languages?

TABLE 3.
Mean JAS Scores of Hospitalized Male CHD Cases and Hospitalized Controls, Bridgeport, Connecticut

JAS Scale	Hospitalized CHD Cases N=40	Hospitalized Controls N=29	t-test	P*
Type A	3.16	−2.73	2.49	.008
Speed & Impatience	0.67	−3.30	1.49	.070
Job-Involvement	−7.99	−6.96	−0.39	.348
Hard-Driving	12.60	4.40	3.09	.002

* One-tailed probabilities

Let's turn our attention to eastern Europe and ask whether Type A behavior exists in socialist countries? Such societies organize their occupational hierarchies, reward systems, and governmental structures differently. We worked with a Polish colleague, Dr. Kazimierz Wrzesniewsk, to develop a Polish translation of the test. Polish-Americans in Boston back-translated the test into English, and then we made a substantial number of changes because the first translation didn't really catch subtleties in meaning. We made the needed changes and sent it back. The Polish team administered the revised form to CHD cases at the Inowroclaw Cardiac Rehabilitation Center and a healthy control group recruited from two factories. The CHD patients were successive cases tested in groups as they came through the Cardiac Rehabilitation Center. The controls were selected to be equivalent in age and occupational level. It was found that the mean Type A score was 3.6 — which is quite similar to the means found in U. S. samples of CHD cases. The mean for the controls was close to zero; significantly different from the CHD cases. The Speed and Impatience score was not remarkably different between the two groups. Job Involvement differences were again in the "wrong direction," but the Hard-driving, Competitive score was very strongly related to CHD at P = .01 (See Table 4). So even in a socialist country with a Slavic language, the Type A and Factor H scores are strong discriminators of CHD cases. The consistency of the job involvement factor in not being positively associated with CHD most of the time suggests that Job Involvement per se may not be a deleterious component of the Type A pattern. However, the Hard-Driving factor and the clinical composite scale of Type A manifestations are better indices of those influences on the central nervous system which are associated with increased risk of coronary disease.

A description of these scales is in order. The Type A scale was developed by discriminant function techniques to best duplicate the clinical judgment that goes into the Structured Behavior Type Interview. The three remaining scales were derived by factor analysis. The items that were valid discriminators between Type A's and B's were put into a factor analysis which yielded three independent dimensions, uncorrelated with each other but all parts of Type A.

To this point, we've presented a variety of retrospective studies from the U. S. and abroad, but as Dr. Kuller pointed out, these are subject to all kinds of biases. Maybe the Type B's have a high case fatality ratio, so when you conduct a study of an out-patient clinic 4-6 weeks later, the Type B's are no longer with us and we have a residue of Type A's taking these tests. Maybe people change from B to A or change in some other peculiar ways after having an MI, so the retrospective test would be open to some form of retrospective bias. The only way to overcome these limitations, of course, is to do a prospective study. This was done as part of the Western Collaborative Group Study, and the results are presented in Table 5. 2,750 healthy men took the test in 1965. Over the next four years, 120 individuals developed coronary disease. From the remainder we excluded the prevalence cases and also a handfull of suspect cases on which the cardiologists could not agree whether the man was a case or not a case. Of the remainder, known to be free of CHD, a 20% random sample was drawn and entered into this analysis. We see the Type A score is not as high in these incidence cases as it was in some of the prevalence case samples, but it is still significantly higher than the mean for the healthy men at the P = .01

TABLE 4.
Mean JAS Scores of Male CHD Patients 8-10 Weeks Post-Infarction Compared With Men Free of CHD, Inowroclaw, Poland

JAS Scale	Myocardial Infarctions N=149	Healthy Controls N=88	t-test	P*
Type A	3.60	0.58	2.47	.007
Speed & Impatience	2.91	2.00	0.73	.233
Job-Involvement	−4.26	−2.50	1.53	.063
Hard-Driving	7.16	3.28	2.86	.003

* One-tailed probabilities

TABLE 5.
Means of JAS Scores in the WCGS for Prospective CHD Cases and Control Group

Scale	t	CHD Cases† (N=120)	Controls† (N=524)
Type A	2.30*	1.70	−0.60
Speed and Impatience	0.28	0.47	0.18
Job Involvement	−0.46	−0.45	0.03
Hard-Driving	0.38	0.18	−0.19

* P=0.01 (one-tailed probability)
† The standard errors of these mean differences are 1.00, 1.02, 1.04, and 0.98 respectively.

level. None of the other scales are significantly discriminating. In the prospective situation only the Type A scale is predictive, but it is statistically predictive of future coronary disease among men who were healthy when they took the test. When we categorize people by their JAS score rather than by their case/non-case status, we observe the relationship shown in Figure 1. Men in the top third of the Type A score distribution had 1.7 times the risk of coronary disease as the men in the lower third of the distribution, which is not as strong as is required to make clinical predictions. (One would like to see relative risk ratios of 3 or 4 to 1.)

Many of the traditional CHD risk factors generate relative risk ratios from 1.7 to 3.5. Considering the primitive nature of behavioral measurements, this is an encouraging beginning. Nevertheless, one cannot take a high score and tell a man that he has a high risk of coronary disease on the basis of the JAS score or interview alone. That would be a misinterpretation and an erroneous way to use these scores. I think these scores, if high, do add more information to the data on other risk factors and help round out the clinical picture of risk, but they are too insensitive to be used by themselves as predictors of risk.

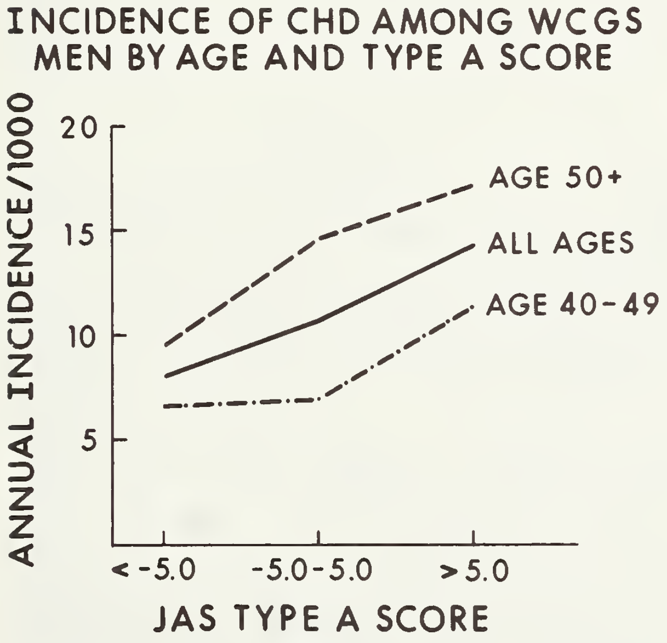


Figure 1

To this point we have looked only at the risk of coronary disease by Type A without controlling for risk factors. But there is much other medical information we ought to take account of — blood pressure, cholesterol, smoking, obesity, and the like. Could these be higher in Type A persons and account for their observed excess in CHD incidence? Let us re-analyze the Western Collaborative Study 4½ year incidence data. We studied Type A ratings by interview in this instance, and controlled statistically first for cholesterol, then blood pressure, and finally for many other variables, one at a time. It was found that Type A was a valid predictor of coronary disease even when each of about a dozen variables was controlled statistically. Then, the skeptic might argue that maybe it wasn't just individual traditional risk factors but rather the interaction of several of them that would eliminate the predictive importance of Type A.

We decided to address this complex explanatory hypothesis and calculated a multiple covariance analysis involving 12 variables. These are listed and the results are shown in Table 6. With all these other factors controlled, it was still found that behavior type was a predictor of whether or not a person became a CHD case or not. For men ages 39-49 years at intake, behavior Type A had adjusted rates per year of 8.9 per thousand, the Type B, 4.50 per thousand, a little better than 2 to 1 ratio. In older men ages 50-59

TABLE 6.
CHD Incidence in Behavior Types A and B with Twelve Other Risk Factors Controlled By Regression

Men aged 39-49
Regression analysis

Source	d.f.	Mean square	F	P
Within cells	2226	0.027		
Regression on 12 variables*	12	0.123	4.60	0.001
Behavior type	1	0.265	9.93	0.002

Adjusted CHD rates: Behavior type A: 8.9/1000/yr.
Behavior type B: 4.0/1000/yr.

Men aged 50-59
Regression analysis

Source	d.f.	Mean square	F	P
Within cells	902	0.066		
Regression on 12 variables*	12	0.251	3.79	0.001
Behavior type	1	0.134	2.02	0.155

Adjusted CHD rates: Behavior type A: 18.7/1000/yr.
Behavior type B: 13.2/1000/yr.

* The 12 variables are as follows: Age, serum cholesterol, beta/alpha lipoprotein ratio, lipalbumin, systolic blood pressure, diastolic blood pressure, number of cigarettes currently smoked per day, Ponderal Index, rating of physical activity on the job, rating of amount of voluntary exercise, amount of schooling, level of income.

years, the ratio is only 18.7 to 13.2, about one and a half to one, after the other 12 variables were taken into account. Although the behavior type was no longer statistically significant in this older group, there was still a gradient. It appears that behavior type is a stronger risk factor in the younger man than in the older man, at least from this analysis.

The next question we want to raise is whether behavior type is related to all clinical forms of coronary disease. Is it related to angina and silent myocardial infarction, as well as to acute infarction? When we divide individuals by the presence of symptomatic myocardial infarction, unrecognized or silent infarction and angina pectoris without infarction, the interview type A's have about twice the incidence rate of the interview type B's in each of these instances. For all three of these forms of disease, there is significant prospective relationship. The importance is that this broad relationship does not seem to hold for anxiety, depression and neuroticism. These factors seem to be prospectively related to angina pectoris, but not to myocardial infarction.

Another way to control for other risk factors is through the multiple logistic function. Professor Richard Brand, biostatistician at the University of California School of Public Health at Berkeley, performed these analyses. When the population in Western Collaborative Group Study is rank ordered by decile of risk calculated from the Framingham risk factor equation, Type A's have much higher rates of coronary disease than Type B's, at each decile of standard risk factors.

Perhaps of even greater value to clinicians is the question of what can one do after a patient already has the chance of getting a second infarction? It is, of course, the finding that provides a justification for attempting clinical intervention.

The next study involved men who survived their first heart attack. There were 287 of them in the Western Collaborative Group. Of this group, 67 developed a recurrent MI and 220 remained with only the one attack over the period of the followup. We examined the standard risk factors to see if any of these would give us a clue to what was related to the risk of having a second MI. In this particular age bracket and group, we found no difference in age, triglycerides, or diastolic blood pressure associated with occurrence of a second MI. Serum cholesterol level was significantly higher in recurrent cases and the number of cigarettes smoked was quite different. It is remarkable that even the people with MI were continuing to smoke at a relatively high rate. It can be seen in Table 7 that Type A was strongly related, with recurrent MI cases having the high mean of 3.96. The JAS factor scores were not related to recurrent MI. Thus, this study found three variables associated with recurrent MI: Cholesterol, number of cigarettes, and the Type A's. These and other standard risk factors were allowed to enter a discriminant function equation. The computer program selected predictors of recurrent MI one at a time in the order of their discriminatory strength. Type A score entered first with a very strong F-value and statistical significance. When other predictors were controlled statistically for the presence of Type A in the equation, the number of cigarettes smoked remained quite strongly related. When cigarettes were included in the equation, the cholesterol level, by reason of its correlation with cigarettes, became less important statistically and entered at only a marginally significant level. (See Table 7.) Type A alone enabled correct classification of 58% of both cases and non-cases. Adding cigarettes as a predictor raised that to 62-63%. Further adding cholesterol did not improve the classification table. In this population and for these variables, Type A seemed to be the strongest predictor of who went on to have a

TABLE 7.
Stepwise Discriminant Function for Classification of Single and Recurrent CHD Events *

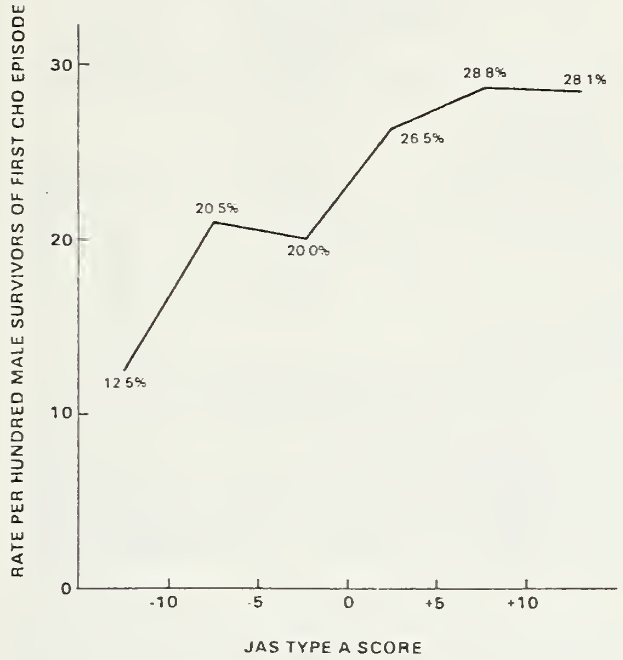
Step No.	Variable Entered	Means** Single Event	Means** Recurrent	F-value To enter	P	% Correctly classified Single Event	% Correctly classified Recurrent
1	Type A score	.18	3.96	7.61	.01	58.3%	58.3%
2	No. of cigarettes	10.49	14.98	4.61	.04	62.3	63.3
3	Cholesterol	242.18	255.42	2.93	.10	62.7	60.0

* Prior probabilities set at .50 for each group.

** This analysis is based upon only those subjects having data on all variables thus reducing the sample sizes to 205 and 60.

Figure 2

RATE OF RECURRENT MYOCARDIAL INFARCTION PER HUNDRED MALE SURVIVORS OF FIRST CHD EPISODE (AGES 39-59 YEARS) ARRAYED BY TYPE A SCORE



second infarction. We can show this relationship in graphic form (Figure 2) when people are classified by their JAS Type A score into approximately equal sixths of the population. The risk of a second MI goes up in curvilinear fashion, rising by the level of the Type A score, in a dose — response relationship.

Doctor Kuller also made mention of the studies using the newer technology of angiography. The Boston University Medical Center has a team of cardiologists doing large numbers of angiograms. They rated these in terms of percent of luminal obstruction in each of four specified coronary arteries. Independently we obtained a series of psychological tests covering anxiety, depression, a variety of neurotic tendencies, and Type A behavior. Ninety-five men who underwent coronary angiography for a wide variety of clinical reasons were divided into men who had zero or one vessel blocked 50% or more, and those who had two or more vessels blocked 50% or more. Among the low atherosclerosis group were some men with one vessel affected 50% or more. Perhaps we shouldn't have included them in the comparison group because they had atherosclerosis too. Nevertheless, we needed to include them or the sample size would have been too small to do a strong statistical test of the hypothesis. In addition, the comparison group included men with valvular disease, chest pain of undertermined origin, cardiomyopathy, and rheumatic heart disease.

Table 8 shows that the people with lesser obstruction of the vessels were clearly Type B, and those with serious obstructions were clearly Type A, as the differences were quite strong. In addition, all three factor scores on the JAS distinguish the group significantly. The men with greater atherosclerotic deposits in their vessels scored much higher on the speed and impatience scale and hard-driving scale. All of the sample tended to be Type B on job involvement but the more atherosclerotic group was closer to Type A than the less obstructed group. This study is cross-sectional and hence we don't know whether the disease or behavior pattern came first.

TABLE 8.
Mean JAS Scores of Patients (by Number Coronary Vessels with Luminal Obstruction $\geq$ 50%)

JAS Scale	Zero or One Vessels Affected (N=37)	Two to Four Vessels Affected (N=55)	F-Test Prob. *	
Type A	−2.45	2.96	7.11	.005
Speed & Impatience	−1.18	2.64	3.05	.04
Job-Involvement	−8.54	−4.37	4.09	.03
Hard-Driving	3.95	8.60	3.69	.03

* One-tailed Probability

Table 9 displays the means of the anxiety and neuroticism scales which were administered at the same time. These are MMPI-type scales. Anxiety and depression scores were significantly higher in men with more severe disease. The hypochondriasis and hysteria scales from the MMPI were not related. The hysteria score has two subscales within it. They are called "admis-

TABLE 9.
Mean Scores of Anxiety and Neuroticism Scales (by Number of Coronary Vessels with Luminal Obstruction $\geq$ 50%

MMPI Derived Scale *	Zero or One Vessles Affected (N=37)	Two to Four Vessels Affected (N=58)	F-Test Prob. * *	
Anxiety (Bendig)	48.1	53.1	5.64	.02
Depression (Dempsey)	51.8	57.9	10.52	.002
Hypochondriasis	59.3	62.0	1.49	.23
Hysteria	64.8	64.6	0.01	>.50
Admission†	59.1	62.1	1.48	.23
Denial†	58.8	55.1	3.96	.05

* Minnesota Multiphasic Personality Inventory (MMPI).
** Two-tailed Probability.
† Sub Scales of Hysteria.

sion of symptoms" which is very much like the hypochrondriasis scale, and "denial of interpersonal problems" reflecting the bland indifference associated with hysterical patients. We found that people with greater atherosclerosis had lower scores on denial. This runs counter to the hypothesis of Hackett and others that people with coronary disease are strong deniers. However, we found subsequently that this denial scale does not correlate with Hackett's clinical judgments among a group of his patients at Massachusetts General Hospital Hence, this denial scale measures a construct quite different from that which Hackett's denial interview measures. Our findings suggest there may be a breakdown of defenses in those people with more severe coronary diseases that is consistent with their higher scores on depression and anxiety.

TABLE 10.
Two-way Analysis of Convariance Determining

Probability Values for Differences Among Means of Scale Scores for Male Patients Grouped by Levels of Two Cardiovascular Pathologies

	Main Effects[1]	
Scale	**Diseased Vessels[2]**	**Angina Intensity[2]**
Type A	.02	---
Speed & Impatience	---	---
Job-Involvement	---	---
Hard-Driving	.04	---
Denial (low)	.01	---
Anxiety	.01	---
Depression	.001	.06
Hysteria	---	.002
Hypochondriasis	---	.02
Admission	---	.01

[1] No interactions were significant and hence main effects are directly interpretable.

[2] Each main effect statistic eliminates the impact, if any, of the other main effect, and removes by covariance the effects attributable to age and number of prior myocardial infarctions.

Our next concern was about the influence of angina pain on the way people respond to questionnaires. Perhaps the people with more severe vascular disease would be experiencing more anginal pain which might influence their responses to items about anxiety, depression, or even Type A behavior. We therefore controlled statistically for the angina intensity which was reported. Then we partialled out the influence of age and the number of prior myocardial infarctions, and with these things controlled, looked at the relationship between these scales and the number of diseased vessels. Type A score was again significantly associated with the number of diseased vessels. Two of the factors dropped out, but the Hard-driving score remained significant, as did anxiety, depression, and low denial. All these scales were associated with diseased vessels even when the reported intensity of anginal pain was taken into account statistically. (See Table 10.) Then we looked at angina intensity and asked if there were some psychological scales which could predict angina intensity apart from the amount of disease in the vessels. In other words, how much of the angina experienced was coming from these persons' psychological makeup rather than from the morphologic limitations of the blood flow to the heart. We found that the scales measuring depression, hypochrondriasis, hysteria and admissions of symptoms were all related to angina intensity after the amount of disease was eliminated statistically and age and number of prior MIs were taken into account by covariance. These scales appear to be good indicators of exaggerating the amount of chest pain. Notice that none of the Type A scales are predictive of anginal intensity once the amount of disease is controlled. However, the Type A scales are related to the amount of disease in the vessels once intensity of anginal pain is controlled.

The validity of these findings is supported by three other studies which have found that Type A behavior, as measured by the Structured Interview, is strongly related to amount of atherosclerosis. These studies were done at Duke University in North Carolina, Columbia University in New York City and by Dr. Perosio and colleagues in Buenos Aires, Argentina.

Our next step was to perform an item analysis of the MMPI items and the JAS items to see which specific items were associated with advanced vessel disease. We found that the 15 MMPI items associated directly with the amount of vessel damage fell into two clusters. One was a series of eight items dealing with social insecurity and self-depreciation and the other was a series of three items dealing with tension and anxiety. We scored people on the eight items that dealt with social insecurity and classified them both on the number of socially insecure items they had endorsed and on the Type A score, split at the median. Within each cell of Table 11, we determined what percentage of persons had two or more coronary arteries obstructed at least 50%. We found that for the group which had neither of

TABLE 11.
Joint Association of Type A and Social Insecurity Score With Severity of Atherosclerosis

Percentage of Patient Group With Two or More Coronary Arteries Obstructed at Least 50 Percent

Social Insecurity Score

		0-1	2-7
Direction of Type A Score	Type B	25% N=24	68% N=19
	Type A	52% N=21	91% N=23

Modified Minimum Chi Square Summary

Effect	df	x^2	P
Type A	1	5.14	.02
Social Insecurity	1	12.26	.001
Lack of Fit (Interaction)	1	1.03	N.S.

these risk characteristics, 25% had advanced atherosclerosis, and men with either one of the risk characteristics had an intermediate rate of vessel obstruction. However, if they were both socially insecure and Type A, 91% had severe atherosclerosis. Some of this association with social insecurity score is circular in that we did the item analysis on this same population and selected only those items associated with advanced atherosclerosis to be entered into the scale. The degree of relationship will be subject to considerable shrinkage in a replication study. The sample sizes in the Table also show that Type A is not correlated with social insecurity as measured by these 7 items, and that these items had an additive contribution to Type A in predicting extent of atherosclerosis.

Psychosocial factors have long been proposed to be associated with coronary heart disease. Recent evidence supports the relationship of two particular groups of psychosocial factors to coronary disease and atherosclerosis: 1) emotional suffering, depression and anxiety and 2) the Type A behavior pattern, both of which have in common the agitation and perturbation of the central nervous system. □

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CLINICAL RECOGNITION OF THE CORONARY-PRONE BEHAVIOR PATTERN

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The coronary-prone behavior pattern (CPBP) is best assessed by observing the subject in action. This behavior pattern occurs in women as well as men, but due to the fact that CHD strikes men 2 to 6 times more often than women, depending on age, it has been studied much more thoroughly in men. Increased research attention to the CPBP in women has only recently been initiated. For these reasons our descriptions of the Type A pattern will be cast in terms of the masculine gender for sake of simplicity of expression. The same clinical symptoms, however, mark Type A behavior in women also.

Type A behavior manifests itself in values, style of thought, manner of interpersonal relationships, response patterns, gestures and movements, facial expressions, rhythm of breathing, and speed, volume and modulation of speech. The Type A individual will direct himself sharply toward his goals, will gesture with abrupt assertive movements, will speak rapidly with bursts of amplitude for emphasis, and by posture and concentration will show an intensity of involvement and a lack of openness to extraneous stimuli not related to his selected goals. The Type B person is more relaxed and open to the environment. He may accomplish as much work in a given time as the Type A, but his movements are more modulated, his voice is even and unhurried, and he does not appear to be as intensely involved. He is not bothered by small diversions from the immediate goal.

The coronary-prone behavior pattern probably originates from deep-seated foundations of the personality. However, it is judged strictly on the basis of overt behavior. This behavior is usually best seen when an individual is actively responding to things that are important to him or in some way challenge or frustrate him. This is why it is difficult, if not impossible, to ascertain the behavior type of a very sick patient or a person who is bored or has disengaged himself from interacting with his surroundings. Once a person is involved in an occupational task, a competitive game, an opportunity to show skill, or a frustrating situation that he wants to change or from which he wants to escape, a variety of behaviors are emitted which permit judgements about the individual's behavior type.

The following sections list a number of indicators of Type A and Type B persons. The categories and the items are neither mutually exclusive nor exhaustive, but they do give a broad picture of the characteristics of the two poles of the continuum of behavior that signals coronary-proneness.

VALUES

1. The Type A person is conscientious and responsible in an intense, inflexible way. Type A's set high standards for themselves and are critical both of themselves and others when they fail to meet these self-established standards. In David Reisman's terms, they are "inner directed" rather than "other directed."

2. The Type A individual prefers being respected for what he does, whereas the Type B usually prefers to be loved for who he is. This leads to the Type A having to maintain productivity in order to maintain his feelings of self-worth. Type A's feel ill at ease if they relax for too long a period and often arrange a shorter vacation so as to get back to doing "something useful." This is the "Protestant ethic" expressed with a vengeance. It is found in people irrespective of religious affiliation and derives from the need, common in Type A persons, continually to prove one's competence and social worth.

3. The Type A individual has more obvious cravings for recognition and power than the Type

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B, but may deny this when asked directly. The work of David Glass suggests that the "need to control" may be a corner-stone of the Type A personality. Those persons who have a calm, easy-going demeanor until confronted with the threat of some loss of control over the things and people around them and who then overreact with hasty, demanding or aggressive behavior are clearly Type A persons who managed a temporary Type B veneer.

4. The Type A individual is compulsively attracted to competition and challenges. He will often compete against himself when no one else is available, trying to better his own previous "record" either in quantity or speed. The Type B person competes as a means of social interaction. He may also try to win but is not as upset by losing as is the Type A. Type B individuals often engage in pastimes or sports just for the enjoyment of it and, unlike Type A's, may not even keep track of how well they are doing.

5. The Type A subject has few sources of gratification other than his job and competitive avocational pursuits and does not seek additional sources. The Type B individual spends his time in a greater variety of pursuits and derives personal gratification from more varied sources such as family, friends, hobbies, artistic and cultural pursuits, or spectator sports. Either Type A or Type B individuals may participate in civic, political, or community affairs, but Type A is more likely than Type B to be "a man of action" and to seek leadership roles.

STYLE OF THOUGHT

1. The Type A person pursues several lines of thought or action apparently simultaneously, for example, working while eating, calculating or even dictating letters while driving an automobile, reading while shaving, or "doing three things at once" while on the job.

2. The Type A person anticipates what is coming next and begins reacting to it in advance. Psychologists call these acts "fractional anticipatory goal reactions." The Type A has his key out long before he gets to the door, he anticipates what another person will do or say next, he starts answering a question before it has been completely asked. Type B's are not in this much of a hurry. They do one thing at a time, and while they are not usually sluggish, they don't often "jump the gun."

3. The Type A individual appears vigilant in expression and posture. He may look "up-tight" or alert and ready to move. The Type B projects a more comfortable air. Type B's can relax more completely and for longer periods.

4. The Type A subject is compulsive about getting things done, overcoming barriers quickly and having his own way. In contrast, the Type B places a greater value on sociability and hence is less abrasive in interactions with other people.

5. The Type A person is competitive in his approach to everyday tasks. He battles the tasks at hand, whereas the Type B approaches tasks with less overt needs for mastery. The Type A fights the "time barrier." He sees all obstacles as challenges, pushes harder, and does not give up even when fatigued. The Type B is more willing to accept inevitable delays and to "give up" when a problem cannot realistically be overcome. He is less obsessed with "being in command."

6. The Type A individual is a poor observer. He fails to register the details of his physical and social environment which do not serve his immediate purposes. As a result, he often is unable to recollect those parts of his experiences that were not at his self-selected focus of attention. One cardiologist, an art collector, typically asks his new patients whether they noticed any painting in the waiting room. The Type B patients can often describe one or more of these paintings, but the Type A's may not even recall that there were any present. Similarly, the Type A lacks insight into his own style of behavior and into the effect he has on other people. The Type A's often fail to note the extent of their own impatience or their irascible demands on others. Family members and work colleagues of the Type A patient are often more perceptive informants as to his behavior than is the patient himself.

INTERPERSONAL RELATIONSHIPS

1. The Type A person tends to be self-centered, whereas the Type B person is able to show more interest in other people. A corollary of this is that the Type A is a poor listener. His spouse or co-worker may complain that he does not pay attention to what others say to him.

2. The Type A individual cannot stay in the frame of reference of other people if it does not coincide with his personal interest or involve his work. This limitation follows from the egocentric-

ity and narrow range of interest of the typical Type A.

3. Feelings of anger are more easily aroused in Type A than Type B persons. Because many Type A's rigidly enforce high standards of social conduct for themselves, they will struggle to inhibit expression of these hostile and aggressive reactions. A spouse or best friend can often give a vivid description of the anger and resentment a Type A person experiences when delayed or blocked, yet the Type A person himself will often deny the intensity of these feelings.

One cardiologist frequently interviews coronary patients and their wives jointly. He asks the patient, "if a car ahead of you is going too slowly and you are unable to pass, who do you do? Are you in a hurry to pass? Do you honk your horn? Do you curse at him? Do you mutter under your breath?" The patient often describes himself as waiting calmly for an opportunity to pass the slower car safely. However, the wife or other family member often reminds the patient that in fact he does honk the horn, curse, or otherwise become upset by having his pace slowed.

4. The Type A person feels frustrated in his work situation more often than the Type B. He is often upset by the demands of the boss and troubled that the boss does not know the difficulties of the circumstances under which he is working. The Type A also usually feels impatient toward subordinates either for their slowness or the poor quality of their work. Type B employees often find themselves in similarly conflicted circumstances, but their feelings of frustration are not triggered by as many minor problems.

5. The Type A individual often expresses an overt bravado. He exudes a certainty of his own correctness, feels that his capacity is superior both to that of his supervisors and subordinates. He believes that with enough effort he can overcome any obstacle. He also expresses a bravado when facing uncomfortable medical procedures. The Type B is more likely to recognize his own limitations and fallibility. It would seem that in many instances that overt bravado of the Type A really covers a deep-seated insecurity. In occasional situations of repeated defeat or serious emotional drain, the Type A may lose his bravado and reveal the insecurity and feelings of fear that lie beneath the surface.

6. The Type A person may tend to be more aggressive sexually than the Type B, but it is the chase that he loves most rather than the consummation. In hospital wards providing care for coronary patients after their transfer from coronary care units, the Type A patient readily distinguishes himself from the occasional Type B patient by teasing the nurses, telling jokes with sexual implications, and occasionally initiating physical contact.

7. In addition to the demands he makes on other people, the extreme Type A individual is victimized by his own behavior pattern. He usually has no insight into this tendency and overestimates the degree of flexibility he really has. People associated with the Type A individual either are drawn in to become victims of his inflexibility or run the risk of incurring his impatience and resentment. The captivity of the Type A person in his behavior pattern explains why he pushes himself beyond his limits, as through extreme competitiveness..

STYLE OF RESPONSE

1. The Type A person reacts rapidly. When participating in a conversation, the Type A begins his comments without delay. In fact, he will often begin his reply before the other person has completed his question or statement. This interrupting behavior, known as "speech hurrying," increases the tempo of conversation and makes the other participants feel hurried. The Type A speaks with clipped syllables. Verbal flow is staccato, alternating bursts of words with hesitation for thought.

2. The Type A person speaks with an air of certainty and emphasis. He accents words with a burst of volume. In contrast, the Type B person lets his speech flow more smoothly in amplitude and pace, and hence may sound more equivocal in his tome at times. The speaker who "drones on" when talking of things of personal interest is almost never a Type A.

Two recent studies have emphasized the strength of association of voice characteristics with overall behavior pattern rating based on the structured interview. Both studies used highly reliable systems for rating volume and speed of speech from tape recorded interviews. Speed of speech, volume of voice and sudden increases in amplitude (especially with positive consonants) were the strongest predictors found by Schucker and Jacobs. Vocal emphasis, speed of speaking and lack of delay in answering were the strongest predictors in the study by Scherwitz, Berton and Leventhal.

3. The Type A subject usually wastes no words. He speaks directly to the point. Type B's will often use more qualifying phrases, more parenthetical comments, and may circle around the point before getting there.

4. A Type A individual can slow down his speech or writing on request, but it makes him uncomfortable. It is easier for him to speed up than slow down, but the converse is true for Type B's. This principle was incorporated as one item of a performance test battery for the Type A personality developed by Bortner. Subjects were asked to write a phrase at their usual speed, then the same phrase more slowly, and then they were challenged to write it extremely slowly. Some Type A's were unable to go extremely slowly, and those that did often commented on the difficulties they were having. Type B's did this task quite easily.

5. The Type A person often gives anticipatory nods and "ahems" while someone is speaking to him. He also may give short explosive laughs to quickly signal the speaker that he anticipates the point being made. The Type B will listen intently but without needing to communicate that he has received the various points of the message and without appearing to attempt to speed up the other speaker.

6. When questioned about situations that characteristically frustrate or block them, Type A and Type B individuals will respond differently. The Type A often relives the tension or anger that he experienced in the situation while he describes it. He sometimes uses gestures, an angry tone of voice, or facial expression to act out his original response or how he feels he would react if confronted with the situation again. Type B individuals do not get that emotionally or physically involved in their recollection of such events.

GESTURES AND MOVEMENTS

1. The Type A person gestures with tense, energetic movements. Both extensor and flexor muscles tend to be contracted, giving such movements an abrupt quality. The Type B gestures in a "looser" style, in which the gesture starts slowly and trails off. It is not as "clipped" as the Type A's gestures.

2. The Type A individual often clenches his hands or makes fists when tense. He may tap with his closed fist to emphasize a point. The Type B is more likely to shrug his shoulders or make open-handed relaxed gestures.

3. The Type A individual shakes hands with a strong grip, an active movement, and a warm handshake. The "dead fish" or clammy handshake, when it occurs, is more likely to come from a Type B.

4. When sitting or standing at rest, the Type A person is less likely than the Type B to remain still. The Type A will "motorize," tap his feet, drum his knuckles, fidget in a rhythmic manner, play with a pencil, or shake a leg or foot. These motor movements tend to be rapid, rhythmic, and repeated. They give the impression of an overflow of excess energy bubbling out of the body. They are evidence of restlessness and may suggest impatience. These movements are not the usual "nervous tic" and do not usually have the same quality as "nervous fidgeting." The latter has a more uneasy, rambling quality, whereas the Type A movements are more repetitive and constant in their form and more assertive in style. They are especially common when a Type A is waiting for something.

FACIAL EXPRESSIONS

1. The Type A person often flashes momentary tense smiles marked by a lateral pull of the mouth with a tight horizontal lip line. This "grimace" is almost a tic in the extreme Type A individual. In contrast, the Type B person takes the time to generate a full smile and, because of more relaxed facial muscles, has more curve to his mouth and fuller lips.

2. The Type A individual flashes brief smiles to give emphasis to comments and to reveal impatience. The smile of the Type B is a more spontaneous expression of open feeling.

3. The Type A person sometimes tenses his jaw muscles or grits his teeth when emphasizing comments. This gives rise to clipped speech. The Type B does not hit consonants with such clipped distinctness. Facial muscles are more relaxed.

BREATHING

1. The Type B person breathes with a regular rhythm that is not serioulsy disrupted when he engages in conversation. The Type A has more frequent breaks in breathing rhythm, heaving

sighs or taking occasional very deep breaths during conversation.

2. The Type A person will frequently inhale more air than necessary while hs is speaking. In the middle or at the end of a sentence this air will be released as an expiratory sigh. This type of sighing is not readily noticed unless the observer is particularly keyed to look and listen for it. It is not the same kind of sighing observed in depressed patients, the latter being slower and less pressured. The prominence of irregular breathing and sighing was first noticed on tape-recorded interviews with Type A individuals.

3. This pattern of irregular breathing and expiratory sighing suggests that the Type A person may be acting as if he feels under pressure or in an emergency. Apparently the activity of the sympathetic nervous system in this respect is substantially different from that prevailing in Type B's, whose breathing is more regular and in whom expiratory sighing is usually absent.

It will be noted that these seven categories of signs and symptoms of the Type A behavior pattern relate primarily to observable features of the style of response. One must also take into account the content of statements made by the person. Specific kinds of content indicative of the coronary-prone behavior pattern include indications that one is "always on the go," is exceptionally competitive, has high ambitions, demands high achievements of oneself, has a past history of promotion and recognition, has a past history of leadership in competitive situations, is always prompt, is always in a hurry (even when it is not necessary), is impatient and cannot stand to wait, is an aggressive struggler, is deeply committed to job or profession to the neglect of other aspects of life, is serious and precise about performance in all settings. The presence of this "content" can be determined through standardized interviews, checklists or questionnaires.

It should be emphasized that *no* one Type A individual manifests all the characteristics listed in these pages, and even a slow Type B person will show a few. It has been shown that Type A is not a unitary concept or single dimension statistically but that it comprises at least three independent factors that correlate with the total pattern but not with each other. Whether or not a person is judged Type A clinically depends on an intuitive summing of the number of characteristics possessed and their intensity. There is very little evidence as to whether individual items in the preceding list are associated with coronary disease but there is much evidence, cited earlier, that the coronary-prone behavior pattern as a whole is predictive. The reason for including these signs and symptoms at much length is the clinical experience of Rosenman and Friedman, conveyed in their collaborative work, and my own further experience in studying the coronary-prone behavior pattern both clinically and statistically.

What are the possible mechanisms by which Type A behavior exerts its pathologic impact? These will be dealt with at greater length elsewhere in this symposium, but I will touch upon a few mediating mechanisms which I believe to be important. First, the Type A seems to have more labile physiological responses. Among Type A's put into an experimental situation, heart rate and blood pressure are more variable than Type B's put into the same situation. There have been studies showing that platelet adhesiveness changes differently as a function of exercise in Type A's and Type B's. Some of Dr. Meyer Friedman's work on norepinephrine secretion suggests that this may be a very important mechanism by which Type A exerts itself. Type A's seem to secrete more epinephrine during active daytime hours than Type B's, although the night time secretions are not different. The resting serum cholesterols of Type A and B do not seem to be particularly different, unless you are dealing with very active Type A's and rather lethargic Type B's. It has been found in a number of studies in Europe, Canada, and the United States that higher cholesterol levels are associated with activation and mobilization of energies, but not with heightened anxiety. In chronic high anxiety states, it appears that cholesterol levels go down. In agitated depressions, however, cholesterol levels have sometimes been found to be elevated. In anticipation of important demanding tasks, cholesterols go up. This has been found in medical students anticipating major examinations, in accountants, as has been mentioned before, and in Navy "frog men" about to have their final examination on underwater demolition. Anxiety seems to affect free-fatty acids, but it doesn't seem to affect cholesterol.

An important point to keep in mind is that the coronary prone pattern, and most other psychosocial risk factors, operate independently of standard risk factors. In addition, it has been shown that Type A and other psychosocial risk factors are operative even when cholesterol, blood pressure, smoking and other traditional risk factors are statistically controlled. Frank's study of coronary angiography (JAMA 1978) showed that Type A was related to amount of atherosclerosis even after blood pressure, cholesterol and smoking history had been controlled statistically. The coronary-prone pattern is also a risk factor for atherosclerosis in the peripheral arteries. Adler and colleagues in Switzerland have reported that severe atherosclerosis of leg arteries is associated with the Type A behavior pattern.

It also appears that different psychosocial factors from the ones discussed above influence hypertension and still a different set may influence sudden death. In a study of air traffic controllers which was just completed at the Boston University School of Medicine, it was found that the air traffic controllers who later were to become hypertensive had lower Hard-Driving scores on the JAS than the ones who remained normotensive. Low life change and low Hard-Driving scores were risk factors for future hypertension in this sample. This is contrary to the picture usually reported for coronary disease.

An interesting study of sudden coronary death by Weinblatt and others at the Health Insurance Plan of New York was published in NEJM in July, 1978. It showed that low education potentiates the risk of complex ventricular premature beats and raises the rate of sudden coronary death.

Unpublished analyses of the WCGS data suggest that Type A is not specifically a risk factor for sudden coronary death. Type A's did have higher coronary mortality rates, but this was due to the fact that they had more infarctions and not because of a higher case fatality ratio for first infarctions.

What are the implications for intervention? There is no proof from field trials, as yet, that intervention on psychosocial risk factors will necessarily reduce CHD or coronary death. This needs to be studied empirically. Clinically, it certainly would do no harm to reduce the intensity of the Type A pattern. Certainly we can reduce the hurry sickness that people have and reduce the intensity of competitiveness and aggressiveness. Let Type A's and B's alike continue their work, continue having high standards, continue trying to achieve, but with less hurry and less struggle. We need to reduce the need for complete control of one's circumstances and of one's environment and hence, reduce the need for Type A's to struggle constantly against the natural resistance of the environment. This may reduce coronary risk, but certainly it should improve the quality of life. There are no negative side effects to this type of intervention.

How is this intervention approached? By changing the basis of self evaluation from "what I do or what I produce" to "what I am." We should look at our intrinsic worth based on qualities and virtues, not quantities. To check basic values we can have our patients ask themselves — and we can ask ourselves: What are the really important things in life? Are they more things, more prestige, achieving ever higher goals? Or are they qualities of character, congenial friendships, mutually supportive family relationships, enjoyment of the good things in life: art, music, literature. There is quite a distinction between Type A's and B's in this respect. We might also encourage our patients with coronary disease who are Type A to foster interest in activities: both productive and leisure time activities involving esthetics, relaxation, non-competitive athletics, and rendering personal and civic services of a type not oriented toward gaining power or prestige. Environmental changes are also an important approach to changing the Type A behavior pattern. When we remove the triggering stimuli that bring out the Type A behavior, we can remove some of the behavior itself. That's why I gave up golf many years ago. I did so poorly and got so furious and so frustrated about it that I put the golf clubs in the attic and eliminated a lot of "striving without joy" as well as much Type A behavior.

There are also implications for intervention to reduce other types of psychosocial risk factors. Physicians would seem well advised to deal actively with sleep problems in patients with high CHD risk because of their apparent contribution to risk of MI. Chronic emotional drain, depression, exhaustion should be dealt with. I recently received a manuscript from Maastricht, Holland, showing that a questionnaire dealing with emotional drain, depression, and "giving up" was associated with future MI's. The questionnaire had items on it like "I feel like I'm a battery that's running out of energy," "Life is not as good to me as it used to be," "I sometimes feel like giving up," "I'm tired all the time."

The intervening clinician, working with a coronary patient or a high coronary risk patient, should search for solutions to chronic conflict and life dissatisfaction situations which create sustained anxiety, either by changing the circumstances, like getting a mother-in-law to move out of the house, or by changing the attitude toward the situation, "mother-in-law's peccadillos are really not important to me so that I need to feel continually upset." Another possibility for psychosocial intervention is to provide emotional and social support during upsetting life crises.

Of course, the reduction of standard biological risk factors like smoking, serum cholesterol, or elevated blood pressure, is particularly important in people with psychosocial risk factors, because these risk factors all appear to be additive. Any reduction in standard risk factors is thus very helpful in lowering probabilities of CHD, particularly where psychosocial risk factors are intense and are unresponsive to intervention. ☐

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QUANTITATION OF PHYSIOLOGICAL RESPONSE TO EMOTIONAL STRESS

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Current interest in the relationship between emotional stress and cardiovascular disease is based upon considerable evidence implicating a wide variety of occupational, social and physiological factors in the pathogenesis of coronary artery disease, arrhythmias and sudden death. Scientifically, the most convincing evidence emanates from very carefully controlled animal experimentation. This body of knowledge was described in great detail by Dr. Henry earlier in this conference.[1] Unfortunately humans do not appear to respond in as pathophysiologically consistent a manner as animals. Humans manifest a large amount of inter-individual variability in response to stress, primarily because of differing perceptual appraisal of the threat and varying coping capacities. Therefore, much additional clinical work on human populations is needed.

Quality control of these factors in human stress experimentation is essential. Highly sensitive, objective and comprehensive measures of human physiological strain responses are needed. Furthermore, the external stressor must be standardized so that it can be utilized in a wide variety of experimental settings. It would also be desirable to include "real-life" relevance and a convincing argument regarding the importance of the stressor situation.

The purpose of this paper is to present a review of traditional psychophysiologic research relating to emotional stress, to describe a typical clinical study on emotional stress and lastly to discuss a different and relatively new method for analyzing the stress/strain relationship in humans.

EXPERIMENTAL INDUCTION OF STRESS

The majority of the previous research in human populations under stress has been conducted in field studies. Some examples include the empirical observations of individuals in "real-life" stressor situations, such as air-traffic controllers,[2, 3, 4] refugees and military personnel in wartime,[5] accountants nearing income tax deadlines,[6] speakers before a critical audience,[7] students during academic examinations[8] and aerospace personnel preparing for a spacecraft launch.[9] The greatest advantage of the field study is that the individuals being examined are experiencing very relevant stressors. In contrast, the greatest disadvantage is that the experimentor has little opportunity to randomly sample the population or to experimentally manipulate the conditions. Therefore, researchers have gravitated toward laboratory experimentation.

The greatest advantage of laboratory investigation is that the experimentor can randomly select the population and can experimentally manipulate conditions to answer very specific scientific questions. Furthermore, the individuals under study can be monitored continuously with highly sophisticated (though cumbersome) electronic equipment. Obviously, the disadvantage lies in the fact that it is not possible to induce the same "real-life" stressor experiences found in field studies. Laboratory stressors must be contrived by the experimentor and therefore of necessity, for ethical reasons, they must be milder in intensity.[10] For some individuals, the laboratory stressor may be more or less relevant and the experimentor must emphasize the importance of performing well although the test structure incorporates elements of uncertainty of outcome and lack of control. These factors are important to ensure that the individual is exposed to a significant degree of stress and potential strain.

Some of the laboratory stressors used in the past include films of disaster and trauma,[11] ego-threatening IQ type tests,[12] speed and accuracy performance tasks,[13] stressful interviews[14] and situations requiring subjects to lie in a lie-detection situation.[15] A most remarkably threatening stressor is an injection of the drug scoline.[16] It produces rapid paralysis of the respiratory and skeletal musculature for a period of about 100 seconds. The subjects, who are unable to move or breathe during that period often report that they feel like they are dying even though they know they are under careful medical supervision.

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Today, human experimentation committees at all institutions carefully scrutinize all research projects to eliminate threat or risk to the participants promoting the development of a less severe variety of laboratory stressors than the injection of scoline. Some of these include: electrical shock avoidance tasks,[17] non-physical competitive contests,[18] mental arithmetic tasks,[19] the cold pressor test,[20] orthostatic changes in body position,[21] vigilence tasks,[22] and word/color conflict tasks.[23, 24]

PSYCHOPHYSIOLOGICAL EXPERIMENTATION

Cardiovascular reactivity is the physiological indicator of emotional stress which has obvious face validity relative to coronary heart disease. Such reactivity is commonly assessed by observing fluctuations in heart rate and blood pressure. In some of the more innovative "real-life" laboratory experiments, these parameters have been monitored in medical students during written examinations,[25] during a defense or startle response,[26] and in pilots during hazardous missions.[27, 28] In the latter example, the heart rate increase beyond that expected from the oxygen uptake was termed "additional heart rate" and was used as an indicator of psychological activation. Obrist has suggested that heart rate reactivity to stress is a strong predictor of systolic blood pressure lability.[29] Thus, if labile hypertension is a coronary risk factor, it appears to be linked with human response to emotional stress. Other important measures of cardiovascular reactivity include stroke volume and peripheral pulse volume. Williams has demonstrated that peripheral blood flow responds either positively or negatively depending upon the perceptual nature of the stressor.[30, 31] Sensory rejection (mental arithmetic) produced peripheral vasodilation; whereas sensory input (word identification of blurred, scattered letters) produced peripheral vasoconstriction. Future efforts to monitor total peripheral resistance may yield additional important results.

The autonomic nervous system is of great importance in understanding the mechanisms underlying the risk associated with cardiovascular reactivity. The plasma catecholamine response to a specific task demand in coronary prone subjects has been studied extensively.[7, 18, 32, 33] Mental effort with varying degrees of neuro-emotional stress have been shown to be positively correlated with sympathetic adrenal and cardiovascular changes. These are also known to produce various ECG changes (T-wave supression) which may be related to risk of sudden death.[21, 34] Lang has shown that cardiac patients are poor "autonomic athletes."[35] That is, they are unable to control heart rate with feedback. It is apparent that a less flexible, psychologically unbridled cardiac system may increase the risk of cardiovascular illness.

The specificity of response to stressful stimuli appears to be prognostically important relative to the future risk of a variety of stress-related disorders. Rossler has suggested that specific disease syndromes, such as rheumatoid arthritis, are associated with a specific emotion of personality.[36] It is logical to assume that a disease entity such as coronary heart disease might be predicted as likely when specific response patterns are elicited. Okin suggests that the significance of the response may be a function of response magnitude alone.[37] It would be appropriate also to suggest that rapidity of return to baseline might be equally as important as the magnitude and the specificity of response.

Other more traditional psychophysiological measures of arousal or autonomic response have centered around the sweating response. Electrical conductivity across two reference points on the skin is measured. Data are commonly referred to as galvanic skin response, skin resistance and electrodermal response.[38, 39, 40] These are only marginally related to cardiovascular reactivity. In some cases, subjects exhibit a generalized strain response with all parameters being elevated, but in group mean data there frequently is no significant association between cardiovascular and autonomic parameters. These observations indicate a skeptical approach to traditional psychophysiology and suggest that more sophisticated and innovative methods be utilized.

CARDIOVASCULAR REACTIVITY IN CARDIAC PATIENTS

Methodology

Twenty-eight male post-infarct patients were matched according to age and occupational status with an equal number of individuals having no overt symptoms of coronary heart disease. All participants were tested in a quiet comfortable environment within a hospital under the supervision of a physician. Heart rate and blood pressure changes were monitored continuously before, during and after a battery of psychological stressors. The first phase of testing consisted of a 12-minute tape-recorded series of 35 ego-threatening, IQ-type questions designed and previously used by Schiffer and colleagues.[41] After a 4-minute recovery period, participants were asked a series of questions about previous life events of an emotional nature. This procedure was similar to that used by Lown.[42] After another 4-minute rest period, the patients were introduced to the Stroop word/color conflict task.[24] This test usually produces confusion and anxiety because of the conflict associated with reciting colors which are mislabeled. Post-test recovery measures were observed for 6 minutes.

Results of Group Mean Analyses

Group mean and standard error of the mean for heart rate, systolic and diastolic blood pressure are shown in Figure 1. Both groups showed a peak increase in heart rate (8-10 beats/min.), in systolic (15-20 mm Hg), and in diastolic (8-14 mm Hg) blood pressure during the ego-threatening quiz. All of these changes were significant, however, there were no significant differences between groups for any of these parameters. Remarkable differences between groups were noted in the frequency of electrocardiographic changes. Depression of S-T segments and premature ventricular contractions were more prevalent in the patient population during psychological testing. No changes were observed at rest for either group.

The results of the experiment regarding hemodynamic characteristics of post-infarct patients versus normals would appear to be equivocal if no further analyses were attempted. An analysis of group data sometimes overlooks highly relevant individual differences as well as differences relative to initial baseline values. Therefore, it seemed appropriate in this investigation to do further, more innovative analyses of these data. An explanation of the rationale for these analyses follows.

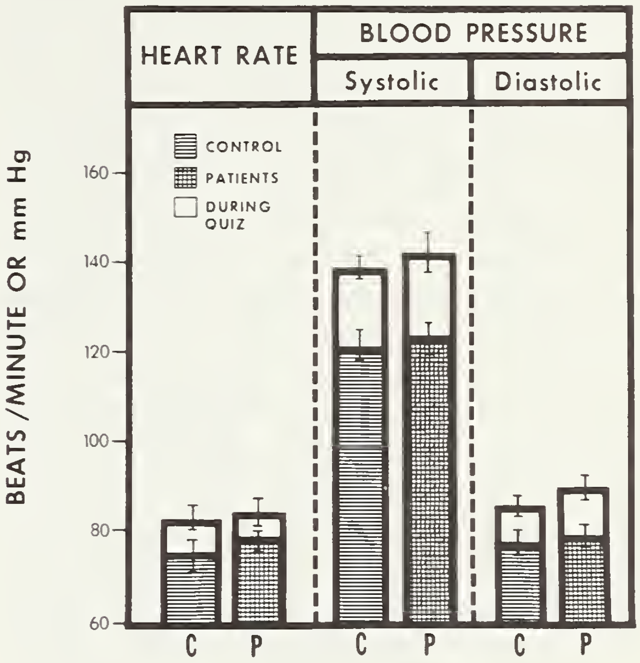

FIGURE 1. Heart rate, systolic and diastolic blood pressure before and during the quiz in two groups — post-infarction cardiac patients (P, N=28) and matched control subjects (C, N=28). Values are mean ± 1 standard error. Values at rest before the quiz (shaded or hatched bars) and average values during the quiz (white bars) are indicated.

RATIONALE FOR THE ELASTIC MODULUS CONCEPT

It is generally agreed that in addition to the steps off observation, recording, analysis, prediction and verification, scientific fields of investigation should include numerical quantitation to be considered truly scientific in the contemporary sense.

Whereas the behavioral field has had some difficulty with the definition and measurement of stress, physical sciences have a more restricted concept of the term. We suggest that analogies with some of the stress concepts employed in physics and in engineering might prove useful for developing quantitative methods for examining behavioral stress. Such an approach is not new and was implied by Walter B. Cannon in 1935.[43] An example of the applications of these concepts will be provided in analyzing the same heart rate data previously described at the conclusion of the paper.

As discussed by Hinkle,[44] in physics and engineering, stress is a highly restricted term confined to the special field of mechanics dealing with elastic properties of solids. In 1676, the physicist Robert Hook observed the effects of tensile forces on materials and noted that the increase in length of a body of material was proportional to the load per unit area applied to the object over a rather wide range of forces. This observation suggested that the amount of increase in length of a body was directly related to the amount of force being applied and became known as Hook's Law. A more mathematically precise definition of stress, consistent with the Newtonian laws of physics, was later developed by the French mathematician Baron Cauchy. Although Hook's original observations involved external loads as stresses and manifest distortions as strain, today stress, strain, and load have specific meanings: Stress is measured in dynes per square centimeter and is the ratio of the internal force brought into play when a substance is distorted to the area over which the force acts; load is the name often applied to the external force producing the distortion, and strain, or distortion, is the ratio of change in size or shape to the original size or shape. In general terms, the relation between these three is stated as Hook's Law: Load = stress = E times strain. The term E is a constant of proportionality which for tensile stress to strain relationships is called Young's Modulus, or the modulus of elasticity. Other elastic moduli exist for the dimensions of volume, hardness, or other dimensional deformities of a material. It should be noted that the concepts of stress, strain, and elastic modulus as used in classical physics and engineering have turned out to be highly special concepts, applicable in practice only to certain classes of solid bodies, only over a limited range and under the assumption that these bodies are entirely homogeneous. The mathematics involved in the precise calculations of stress and strain are more complex than the simple linear equations of Hook's Law would indicate. For purposes of discussion, however, Hook observed that different materials elongate at different rates of change when known loads per unit area were applied to them. In essence, the relationship between stress and strain is relatively distinctive for specific materials and displays a slope or curve. The mathematical description of that curve is called its elastic modulus. In classical physics the elastic modulus is a simple linear relationship.

Disregarding the theoretical and mathematical objections to Hook's Law as a highly constrained scientific application, it is nevertheless evident that the stress to strain relationship for a specific material does display a characteristic slope or curve relating strain as a function of stress or load; and that the morphology of this curve can be mathematically expressed as a simple or complex function of the force applied. Certainly, the stress to strain relationship curve for copper is qualitatively and quantitatively different from the curve for steel. A wide range of materials in the physical sciences are categorized according to the quantitative expressions of their elastic curves. The composite portfolio of such data for a material signifies its identity by recognizing its behavior and also expresses its projected suitability for a variety of structural and stressful functions. A quantity of stress such as 500 dynes per square centimeter is meaningless unless the strength and resiliency of the material under stress is known. By subjecting materials to standard loads and expressing a variety of dimensional responses as strain, one can generate simple tables or graphs which contain discriminating qualitative and quantitative information about the performance capabilities of certain materials and their ability to withstand stress. By analogy, we believe it may be possible to classify and categorize human physiologic responses as dimensional strain patterns under various types of stress.

Although human organisms are more complex than simple material objects, the fact remains that performance characteristics under stress serve as one means of characterizing important attributes of behavior and ability to withstand certain loads and stresses. By way of analogy, measurable physiologic parameters of an individual's behavior might be analogous to the tensile, volume or shear behavioral characteristics of physical materials. A variety of physiologic measurables are dimensional attributes of the organism, are quantifiable, and manifest strain or deviation from the resting state under stress. Examples might include heart rate, blood pressure, or muscle tension. To measure strain in a physiological sense, one need only measure the resting or unstressed dimension and then plot fractional deviations from that value as force is applied to the subject.

The elastic modulus concept is easily applied to physical stress behavior as in exercise testing since workload can be defined in terms of ergs, kilopond meters or other quantitative terms. Furthermore, a graph can be constructed relating physical load to measured strain for a given physiologic dimension (heart rate) and a depiction of an elastic modulus for that physiologic measurable can be created. Within limits, the slopes for such a stress response are mathematically definable and represent the elastic modulus for a specific physiologic dimension of the subject under a specific type of stress. The composite physiologic picture of all elastic moduli for a given subject is one method of developing distinctive and quantitatively categorized expressions of physiologic behavior under physical stress.

Measurement of emotional stress or load requires a seemingly different perspective than exercise testing because external environmental situations appear to be stressful primarily through symbolism and cognition rather than through a direct effect. However, fundamental physical principles tell us that there is no change in the motion of a body (or physiologic function) unless a resultant force is acting upon it. Quite clearly then, the magnitude and direction of any physiologic measurable change must reflect the influence of an underlying force or stress. Considering the complex amalgam of unknown or unmeasurable factors constituting the psychological dimension, it is probably a moot point to attempt to quantify environmental symbolism other than by the response it produces. Since physiologic responses are quite quantifiable, within a standardized psychologic symbolic challenge, the magnitude of strain response for any physiological function must reflect an underlying stress as a cause. Therefore, such strain responses provide comparative identifying information regarding the psychophysiological elasticity of the subject under study.[45] Assigning units of stress magnitude for a psychological stress test protocol is totally arbitrary. The important aspect is that the testing procedures be standardized and uniformly applied to all subjects, and that a reference point be defined for the basal value of the physiologic parameter under study so that strain may be calculated.

Even within the physical sciences, the units of standard physical measurement were arbitrarily contrived, but have proven to be very functional. The usefulness of a measurement tool first requires that the term be precisely defined and expressable in multiple units relative to an arbitrary but standard reference. Provided that a group of individuals are subjected to identical experimental input in a controlled psychological stress test, the differences in physiologic dimensional responses, expressed as strain and plotted against the time course of the test paradigm, can yield important discriminating information in terms of the slopes of response. Like the stress to strain, or elastic modulus expressions of unknown physical materials, psychophysiological stress test curves are one means of mathematically describing and quantifying patterns of psychophysiologic behavior under the conditions of the test paradigm. Although only the external factors of the psychologic stress paradigm can be standardized, differences in strain responses must reflect differences in psychophysiologic cognitive integrative and end organ responses, and are certainly quantifiable in terms of physiologic strain. Therefore, within the confines of the test procedure, such an approach might logically be expected to provide simple and discriminating signatures of individual patterns of behavior and perhaps, for susceptibility to disease.

The Elastic Modulus Concept Applied to Human Response:

A discussion of the application of this concept can be demonstrated by using the heart rate response of the test subjects previously discussed. Note that comparing post myocardial infarction patients and normal subjects, the heart rate response to physical exercise stress results in slightly greater strain per work load for cardiac patients as compared to normal subjects (Figure 2). Strain is defined as the change in heart rate divided by the resting heart rate times 100 (HR work − HR rest ÷ HR rest × 100). Thus, the cardiac subject has a slightly greater magnitude of heart rate strain for a given workload than the normal individual. Conversely, the functional workload capacity (METS) is higher in the normals than in the cardiacs. One can easily see that the cardiac group manifests a more flattened slope in the dimension of heart rate strain and is more easily distorted in this dimension.

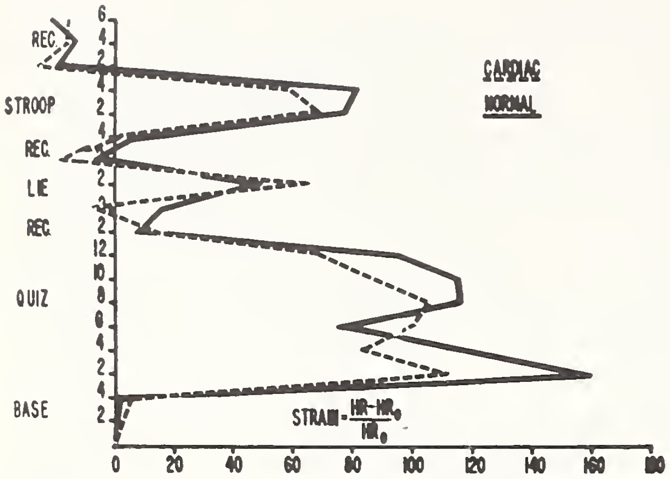

FIGURE 2. Heart rate response to physical exercise stress results in slightly greater strain per workload for post-infarction patients as compared to normal matched control subjects.

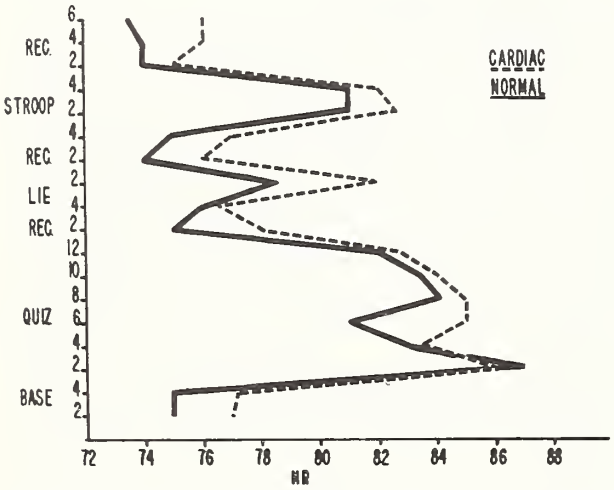

FIGURE 3. Comparison of heart rate responses of cardiac patients and normal subjects to a battery of psychological stress tests — Quiz, Life Inventory of Events (LIE), Stroop Color/Word Test. Cardiac patients — broken line; normal subjects — solid line; REC — recovery.

Figure 3 shows the heart rate response in both groups across the entire battery of psychological stress tests (Quiz, Life Inventory of Events, Stroop Color/Word Test). Althouth heart rate was slightly higher at rest for the cardiac patients, there were no significant differences between the groups. However, when the same heart rate data was computed in terms of strain response, certain differences became evident (Figure 4). The cardiac patients showed an earlier and greater magnitude of depression of heart rate response. Both groups showed heart rate acceleration throughout the first two minutes of the quiz followed by a tendency to return toward basal heart rate, but with earlier acceleration seen in the cardiacs on the rebound phase and more marked and earlier depression during the recovery phase. Furthermore, during the Stroop test, whereas normals continue to accelerate as the task becomes more difficult, cardiacs begin to manifest heart rate deceleration as the test becomes more difficult. In fact, there are two points during this battery of testing in which the directional slopes of the two groups are totally divergent; at 4 to 6 minutes of the Quiz, and from 2 to 4 minutes of the Stroop test.

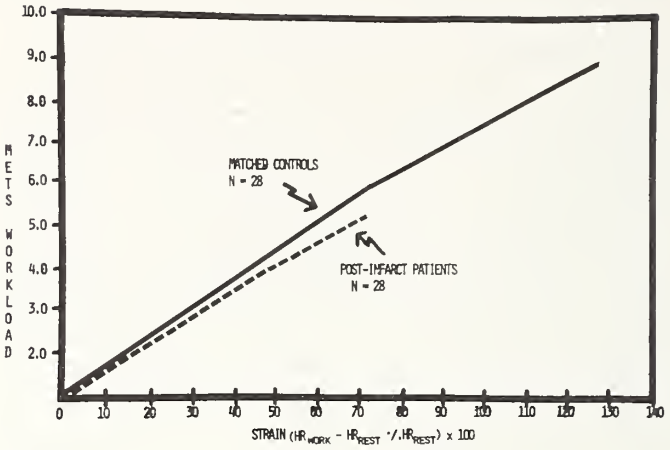

FIGURE 4. Heart rate response data from Figure 3 computed as strain response. Cardiac patients showed an earlier and greater magnitude of depression of heart rate response. See Figure 3 for description of terms.

In conclusion, it appears that the concepts of strain and elastic modulus may be of some use in attempting to investigate and quantify the relationship between emotional stress and human behavior within the area of psychophysiologic stress testing. ☐

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STRESS AND THE REDUCTION OF DISTRESS

HANS SELYE, C.C., M.D., PH.D., D.Sc.*

DEFINITION OF STRESS

First let me express my gratitude to Dr. Robert S. Eliot for convoking this Second National Conference on Emotional Stress and Heart Disease and allowing me to speak to you on this heavenly island about my favorite subject, stress.

I never prepare any manuscript, but I thought, since such was desired, the best I could do is to present an updated summary of my work on stress, started in 1936 and still going full steam ahead.

The word stress, like success, failure, or happiness, means different things to different people: it would seem that no single definition can be agreed upon by the lay public although the word has become part of our daily vocabulary.

There is an important conceptual and semantic distinction which must be made in order to clear up this confusion.

In science, *"stress is the nonspecific response of the body to any demand made upon it."* That is, when an organism is called upon to readjust or adapt in order to maintain normalcy, it is under stress. It is irrelevant whether the agent or situation provoking stress is pleasant or unpleasant: sorrow, joy, heat, cold, worry, pain, trauma, ecstasy — All can induce the physiological manifestations of stress in the body. A "stressor," then, is any stimulus which induces stress. To speak of "emotional stress" or "physical stress" is correct if used to indicate the evocative agent, with reference to different *stressors,* that give rise to the same bodily reactions which are evidence of *stress.* The essence of stress is the demand for adaptation as such, and the intensity and duration of this demand are all that matter.

As is clear from the definition, stress cannot be avoided; no matter what you do or what happens to you, there arises a demand to maintain life and to resist and adapt to changing external influences. Even while fully relaxed and asleep, you are under some stress; your heart must continue to pump blood, your intestines to digest last night's dinner, your muscles to move your chest and thus permit respiration; even your brain is not at complete rest while you are dreaming.

The concept of stress is very old; it must have occurred even to prehistoric man that the loss of vigor and the feelings of exhaustion that overcame him after hard labor, prolonged exposure to cold or heat, loss of blood, agonizing fear or any kind of disease, had something in common. He might also have noticed that whenever faced with a prolonged and unaccustomedly strenuous task — swimming in cold water, lifting rocks, or going without food — he passes though three stages: at first, he experiences hardship, then he gets used to it, and finally he cannot stand it any longer. He did not think of this three-phase response as a general law regulating the behavior of living beings faced with any exacting task. The immediate necessites of finding food and shelter kept him too busy to worry about such abstract concepts as this. Yet, the vague outlines were there, ready to be analyzed and translated from indistinct feelings into the precise terms of science.

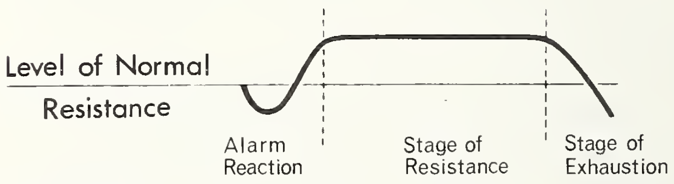

FIGURE. The three phases of the General Adaptation Syndrome.

Alarm Reaction. The body shows the changes characteristic of the first exposure to a stressor. At the same time, its resistance is diminished and, if the stressor is sufficiently strong (severe burns, extremes of temperature), death may result.

Stage of Resistance. Resistance ensues if continued exposure to the stressor is compatible with adaptation. The bodily signs characteristic of the alarm reaction have virtually disappeared, and resistance rises above normal.

Stage of Exhaustion. Following long, continued exposure to the same stressor, to which the body has become adjusted, eventually adaptation energy is exhausted. The signs of the alarm reaction reappear, but now they are irreversible, and the individual dies.

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The name "*General Adaptation Syndrome" or G.A.S.*" was coined to describe the total response of any living being to a stressor. The G.A.S. is comprised of three distinct phases: the alarm reaction, the stage of resistance, and the stage of exhaustion.

The *alarm reaction* represents a "call to arms" of the body's defenses, and is activated when an organism first encounters a stimulus strong enough to necessitate adaptation. Think of the shock of your first dive into a cold lake — even colloquially, it's quite "alarming."

But after a few minutes, the water will feel less cold; you might remark to a less enthusiastic friend that the temperature was really quite pleasant once one became accustomed to it. The *stage of resistance* has been reached, and how long it can last depends upon such factors as the actual temperature of the water, your physical condition, your metabolic rate (e.g., how quickly your body can produce heat necessary to maintain normal temperature), and so on.

But sooner or later, you will probably notice that you are again cold and beginning to shiver. During the stage of resistance, the acquired adaptation to cold is wearing off, and the *stage of exhaustion* is imminent. Physically, you can no longer produce enough heat to offset its loss to the water; in your muscles, the wastes from each cell are beginning to accummulate, resulting in fatigue and aching. A cramp could ensue now, as it could during the shock of the first exposure to the cold.

Only the most severe stress leads rapidly to the stage of exhaustion and death. Most of the physical and mental stressors which act during a limited period produce changes corresponding only to the first and second stages: at first, they may upset and alarm us but then we get used to them. In the course of a normal human life, everybody goes through the first two stages many, many times; otherwise we could never become adapted to perform all the activities and face all the demands which are man's lot.

For instance, a foot race produces a stress situation, mainly in our muscles and cardiovascular system. To cope with this, we first have to warm up and get these organs ready for the task at hand (in doing this, one can avoid the physical extremes of the alarm reaction); then for a while we will be at the height of efficiency in running, but eventually exhaustion will set in. This could be compared with an alarm reaction, a stage of resistance and a stage of exhaustion, all limited primarily to the muscular and cardivascular systems. (In technical terms a "Local Adaptation Syndrome or L.A.S.") But such exhaustion is largely reversible; after a good rest we will be back to virtually normal again.

It is only when the whole organism is exhausted and hopelessly encumbered by the accummulated chemical wastes of life, that one enters into the terminal stage of exhaustion of the G.A.S. This happens in senility, at the end of a normal life span, or through the accelerated aging caused by excessive stress. Only when all our adaptability is used up will irreversible general exhaustion and death follow.

There seems to be a close interrelation between the G.A.S. and aging. People can get used to a number of things (cold, heavy muscular work, worries) which at first have a very alarming effect; yet, upon prolonged exposure, sooner or later, all resistance breaks down and exhaustion sets in. It is as though something were lost or used up during the work of adaptation. Although we have a name for this phenomenon, "adaptation energy," we still have no precise concept of what this energy might be. It is certainly not caloric energy, because exhaustion occurs even if we eat well. It would seem that at birth, each individual inherited a certain amount of adaptation energy, the magnitude of which is determined by one's genetic background; one can draw upon this capital thriftily for a long but monotonous, uneventful existence, or spend it lavishly in the course of a stressful, intense, but more colorful and exciting life. In any case, there is just so much of it, and one must budget accordingly.

Absolute freedom from stress is death. Contrary to public opinion we must not — and indeed cannot — avoid stress. But we can meet it efficiently and enjoy it by learning more about its mechanisms and by adjusting our philosophy of life accordingly.

Man has always tried to identify and neutralize those stressors which could be considered negative, and maximize those which are pleasant. Vast strides have been made in the alleviation of stressors which cause physical discomfort. Much of the world is now clothed, housed, fed and vaccinated. It is natural that in the more affluent West, the main concern for many investigators in this latter half of the twentieth century is to resolve the social and emotional problems with which we are now confronted. The proliferation of alternative mindstyles and lifestyles bears witness to the depth of this concern, with the lay person as well as the professional.

It seems that the most common source of mental or emotional distress is also the oldest: the necessity to choose amongst alternatives. As the existentialists say, "Man is condemned to be free," and libraries are replete with formalized approaches, systems — mindstyles through the use of which one can avoid having to agonize over every situation in life. One need only consult the authority (whatever or whoever it might be) for the correct answer to many of the dilemmas with which one is faced. Of course, for many individuals the choices have already been made and all that is needed is a framework within which to fit these ideas. Others, generally dissatisfied with their lives, are looking for a new orientation, a fresh approach.

Once one's philosophical and moral position is settled, probably the next choice is one of a lifestyle in harmony with it. Once again, one will find many widely disparate ways of life, outlined frequently by their adherents. Of course, the most important, and perhaps the most difficult, choice is always the first one. But almost any direction is better than none at all; as Montaigne put it, "No wind blows in favor of the ship that has no destination."[1]

TECHNIQUES FOR REDUCING DISTRESS

Many therapeutic techniques help us indirectly by improving our physical and mental fitness. Among these, the counsel most commonly given is to relax and refrain from any but the most unavoidable activities, or to seek diversion in leisure and play. However, this kind of advice is more easily given than followed because active people are particularly dependent upon finding outlets for their pent-up energy and cannot relax or play continuously without suffering from the feeling that they are wasting their time.

Work is a basic biologic need of man. The question is not whether we should or should not work, but what kind of work suits us best. In order to function normally, man needs work as he needs air, food, sleep, social contacts, or sex. Few people would enthusiastically welcome the discovery of test-tube babies making sex superfluous; let us not look forward with eager anticipation to the days when automation will make every activity redundant.

For persons with an irrepressible drive to seek stress, it is much easier to find some comparatively unexacting diversionary activity than to do nothing. But because the so-called "leisure occupations" tend to become highly goal-oriented, competitive efforts at outstanding achievement in sports, social, educational and civic events, they represent a poor antidote to the emotional stressors of daily work. This problem is particularly important for those who approach or have reached the retirement age and simply cannot go on utilizing their energy in the way to which they have become accustomed. As the progress of technology increasingly shortens work hours for most standard occupations, semi-retirement will be possible as soon as a career begins. Yet, not everyone knows how to play or to occupy additonal leisure time with the more passive enjoyment of music, painting, sculpture, travel or spectator sports; hence, many people seek to release their pent-up energy through such dangerous outlets as drugs, violence and other forms of destructive behavior.

Why should we work so hard to avoid work? The French philosopher, Henri Bergson, justly pointed out that it would be more appropriate to call our species "homo faber" (the making man) rather than "homo sapiens" (the knowing man), for the characteristic feature of man is not his wisdom but his constant urge to work on improving his environment and himself.

The most important aim of man cannot be to work as little as possible. For the full enjoyment of leisure, you have to be tired first, as for the full enjoyment of food, the best cook is hunger.

Do not listen to the cherished slogans of the agitators who keep repeating that "There is more to life than just work" or that "You should work to live, not live to work." This sounds pretty convincing, but is it really? Our aim should not be to avoid work but to find the kind of occupation which, for us, is play. The best way to avoid stress is to select an environment (spouse, boss, climate, social group) which is in line with our innate preferences, and to find an activity which we like and respect. Only thus can we eliminate the need for constant adaptation that is the major cause of stress.

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[1] Incidentally, in a letter commenting on the British edition of "Stress without Distress," Sir Hans Krebs called my attention to the fact that essentially the same idea had been expressed 1500 years earlier by Seneca (Epistulae at Lucullum, LXXI, 2): "Ignoranti quem portum petat nullus suus ventus est" — "He who knows not his port of destiny has no favorable wind."

Among the procedures we use to protect ourselves against the damaging effects of distress, some are activities employed by the individual as a means of diversion from daily routine, while others are more formal techniques or therapies. The most important among these kinds of treatment are physical or behavioral.

Just as muscular exercise, hot baths, saunas and the like appear to be of some help in combatting the distress of everyday life, a number of psychological methods enjoy considerable popularity. Although these techniques seem to have beneficial effects upon general well-being and mental performance, all cannot yet be fully explained in the understanding of the biochemical changes that accompany the refreshing relaxation induced by such different, indirect means of improving well-being as athletics, relaxed meditation, a cup of coffee, or psychopharmaca (for example, tranquilizers). One must always bear in mind, too, that excessive or inappropriate use of any of these techniques can cause considerable damage, and until we know more about their mechanisms of action, we must proceed with caution.

One of the main causes of distress in modern society arises from the need to make decisions concerning ethical problems. This has always been a troublesome area for mankind, but the situation has become acute with the dwindling credibility and popularity of the traditional bastions of ethical codes, the organized religions. Many individuals now seriously question the relevance of many of their basic tenets, and consequently look elsewhere for a moral basis of their lives.

The instinct for survival and self-preservation is as characteristic of man as it is of other animals and is just as powerful in man. The principal teaching of most religions, to respect the wishes of one's neighbor as much as one's own — "Love thy neighbour as thyself" — is a practical impossibility. While it is, of course, a noble goal, it is one which can never be attained, and furthermore, it is not consonant with man's basic instinct of self-preservation.

It is a scientifically established fact that man has an inescapable, natural urge to work egoistically for things that can be stored to strengthen his own well-being in unpredictable situations with which life may face him. These are not instincts we should combat or be ashamed of. There is no example in nature of a creature guided exclusively by altruism and the desire to protect others. "Love thy neighbour as theyself" is a command full of wisdom, but it is incompatible with biologic laws; no one ought to develop an inferiority complex if he cannot love all his fellow men on command. Neither should we feel guilty because we work for treasures that can be stored to ensure our own future security. Hoarding capital is a vitally important biologic instinct we share with all animals, such as ants, bees, squirrels and beavers.

The philosophy of *altruistic egoism* accepts, as morally correct, egoism and working to hoard personal capital, by the creation of feelings of accomplishment and security through the inspiration in others of love, goodwill and gratitude for what we have done.

The motto "*Earn* thy neighbour's love," unlike love on command, is compatible with man's nature. Although it is based on altruistic egoism, it could hardly be attacked as unethical. Who would blame anyone who wants to assure his own happiness and security by accumulating the treasures of other people's benevolence toward him? This makes him virtually unassailable, for nobody wants to attack and destroy those upon whom he depends.

Man is a social being. One cannot remain alone in the midst of our overcrowded society; one must trust some friends, despite their apparent untrustworthiness, or one will have no friends, no support. If you have earned your neighbour's love, you will never be alone.

My own experience in using this code of conduct has taught me a few little tricks which might help others in the practice of altruistic egoism:

Even if you systematically want to hoard love, don't waste your time trying to befriend an alligator. Admit that there is no perfection, but, in each category of achievement, something is tops: be satisfied to strive for that.

To earn thy neighbour's love, it is not sufficient to help only those who ask for assistance. You must overcome your shyness in approaching people who may desperately need your assistance, but — as so often happens — are too timid or proud to ask for it.

Before each major decision, take time to ask yourself which course will earn you more goodwill. Almost all actions give and take some of it. To give something to one (even if only your time and attention) necessarily deprives another. To offer major assitance that will really be appreciated throughout life, you may have to expose your best friend to what momentarily could seem senseless or purely authoritarian and egoistic behavior. And when you have to communicate an unpleasant decision, the benevolent tone of tact is usually more effective than the cold logic of tactics.

Realize that men are not created equal though they should, of course, have a birthright to equal opportunities. After birth, in a free society, their performance should determine their progress. There will always be leaders and followers, but the leaders are worth keeping only as long as they can serve the followers by acquiring their love, respect and gratitude; or as long as they are useful.

Finally, do not forget that there is no ready-made success formula which would suit everybody. We are all different and so are our problems. The only thing we have in common is our subordination to those fundamental biological laws which govern all living beings, including men. No previous code of ethics — religious, philosophical, or political — has provided specific prescriptions for each possible event that may be met in life. Each individual must deal with a situation in his own unique way, on the basis of his own genetic background, previous experiences, state of health, etc. Other codes of behavior, for example, those of Christianity, communism, or patriotism cannot tell you precisely what to do in each specific situation. Many times you should not "do unto others what you would like them to do unto you," because the requirements of others may not be the same as yours. The decision of how to be useful to different people must remain a problem for your own ingenuity.

The study of the disparate solutions to ways of facing the stressors in life is profoundly interesting in itself; the mere number of mindstyles and lifestyles is proof that no single solution is appropriate to all people. However, to have a basis, a few fundamental, scientifically founded and verifiable criteria can be of help in choosing one's own pathway.

In my introductory chapter to the *Information Please Almanac 1979* I have tried to summarize all this concisely, as follows:

1. *Find your own stress level* — the speed at which you can run toward your own goal. Make sure that both the stress level and the goal are really your own, and not imposed upon you by society, for only you yourself can know what you want and how fast you can accomplish it. There is no point in forcing a turtle to run like a racehorse or preventing a racehorse from running faster than a turtle because of some "moral obligation." The same is true of people.
2. *Be an altruistic egoist.* Do not try to suppress the natural instinct of all living beings to look after themselves first. Yet the wish to be of some use, to do some good to others, is also natural. We are social beings, and everybody wants somehow to earn respect and gratitude. You must be useful to others. This gives you the greatest degree of safety, because no one wishes to destroy a person who is useful.
3. *Earn thy neighbour's love.* This is a contemporary modification of the maxim "Love thy neighbour as thyself." It recognizes that all neighbours are not lovable and that it is impossible to love on command.

In my first book for the layman, I tried to condense several thousand scientific articles and two dozen books into 324 pages — which may still have been too long and too technical. Perhaps two short lines can summarize what I have discovered from all my thought and research:

Fight for your highest attainable aim, but do not put up resistance in vain. ☐

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THE ROLE OF THE CENTRAL NERVOUS SYSTEM IN FATAL ARRHYTHMIAS*

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Epidemiologic evidence from Western Europe and the United States indicate that sudden cardiac death (SCD) due to ventricular fibrillation is the leading cause of fatality in the 35 to 65 age group.[1] In the U.S. alone almost half a million deaths annually are ascribable to this cause alone. In a quarter of these cases, death is the initial and only manifestation of coronary artery disease, the usual underlying cardiac disorder. The problem, largely ignored until this past decade, is now being recognized as a leading public health problem. Contemporary clinical experience indicates that SCD is not the inevitable expression of far advanced atherosclerosis, but represents a reversible and preventable electrical accident. A key question relates to the pathophysiologic factors which trigger ventricular fibrillation. Recent studies suggest the operation of neurophysiologic and psychologic factors as destabilizers of ventricular myocardial electrical stability.

That intimate links exist between brain and heart has been appreciated for centuries and is reflected in the idiomatic expressions of various languages and folk accounts of numerous cultures. Anecdotal illustrations abound of death purportedly due to a variety of emotional states including fear, anger and joy.[2] Cannon[3] speculated on the mechanism of sudden death related to emotional factors. He considered the prototype of this form of fatality to have its roots in so-called "voodoo" deaths resulting from tribal castigation or from superstitious beliefs and fears relating to the occult. Cannon postulated a sympatho-adrenal mechanism as its genesis. Systematic studies in this area, not surprisingly, are lacking. The poverty of investigative effort is related, in part, to methodologic difficulties in quantifying psychologic stresses, emotional responses and physical alterations in the target organ as well as to the fact that "remembered" stress can be deleterious, but is a profoundly subjective state not amenable to perception by the outside observer. Furthermore, the rapid onset of the terminal episode occurring outside of a hospital and its brief duration does not permit appropriate scientific inquiry. Various technologic developments within the last two decades such as the introduction of coronary care units, direct-current defibrillation, cardiopulmonary resuscitation techniques and cardiac rhythm monitoring in the ambulatory patient have helped to provide deeper insights into the SCD syndrome. Coronary care units permit the precise monitoring of heart rate and rhythm in patients with myocardial infarction. Immediate salvage of patients succumbing to ventricular fibrillation is now possible by cardiopulmonary resuscitation and electrical defibrillation. Furthermore, storage of electrocardiographic information on tape and extended periods of ambulatory monitoring in patients deemed at risk for sudden death enable a critical evaluation of ventricular arrhythmias in patients at high risk for SCD.

CONCEPT OF TRANSIENT RISK FACTORS FOR VENTRICULAR FIBRILLATION

Certain observations made in the coronary care unit support the view that SCD in the community is in most instances due to ventricular fibrillation. Some patients recovering from acute myocardial infarction are left with electrically unstable hearts. They may suddenly develop ventricular fibrillation, often preceded by warning ventricular arrhythmias. The resulting disorganized electrical and mechanical activation of the heart rapidly leads to death unless electrical defibrillation is promptly instituted. Since the heart may resume normal beating within seconds after defibrillation and continue to beat normally for many years thereafter, ventricular fibrillation cannot be the inevitable end-result of advanced coronary artery disease. The observation that ventricular fibrillation may occur unexpectedly in an otherwise normally beating heart, and the fact that it can be terminated instantaneously without recurrence suggest that profound compromise of coronary arterial blood flow is probably not the exclusive precipitant of ventricular fibrillation. It is likely, therefore, that ventricular fibrillation is due to transient factors which momentarily disorganize cardiac electrical activity. Thus, a catastrophic arrhythmia may be regarded as an "electrophysiologic accident" which, though almost always fatal when unattended, is frequently reversible by defibrillation.

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An additional significant finding was uncovered in recent studies in patients resuscitated from out-of-hospital ventricular fibrillation. Acute myocardial infarction was absent in two-thirds of patients resuscitated from SCD.[4] Thus, ventricular fibrillation can and indeed does occur in the majority of instances, in the absence of significant damage to the heart. Extensive structural damage is thus not a necessary precondition for ventricular fibrillation responsible for SCD. Based on these considerations, Lown[5, 6, 7] hypothesized that the electrically unstable heart is exposed to periodic risk for ventricular fibrillation from one or more sources. He labelled the inputs responsible for ventricular fibrillation under these circumstances *transient risk factors*. While definitive evidence that transient risk factors operate in man awaits further exposition, it is relevant to discuss the contribution of the central nervous system in the destabilization of normal cardiac electrical activity.

ANIMAL STUDIES

The evidence that transient risk factors derive, in part at least, from the nervous system is most persuasively illustrated by animal experiments. In studies dating back to the early part of this century, a number of investigators showed that CNS stimulation using chemicals, drugs and electrical currents provoked a variety of ventricular arrhythmias including ventricular fibrillation.[8, 9, 10] A number of studies involving stimulation and ablation of sympathetic neural structures demonstrated that the pathway for this response arises in the midbrain and quadrigeminal bodies, traverses the reticular formation, and is finally channelled to the heart via the cardiac sympathetic nerves.[11, 12, 13, 14, 15, 16]

Cardiac electrical stimulation studies during manipulations of sympathetic inputs to the heart have further elucidated the role of nervous influences on vulnerability to ventricular fibrillation. In the normal heart, ventricular fibrillation may be provoked by electrical stimuli discharged during the brief vulnerable period of the ventricle. The minimum current required to provoke ventricular fibrillation is termed the ventricular fibrillation threshold. Stimulating peripheral neural structures such as stellate ganglia in dogs reduces markedly the threshold for ventricular fibrillation, indicating increased vulnerability to ventricular fibrillation imparted by sympathetic stimulation.[5] When the posterior hypothalamus is stimulated, the resulting increase in sympathetic adrenergic outflow also lowers the ventricular fibrillation threshold.[17] Satinsky and coworkers[18] found that coronary artery ligation in dogs resulted in a 6.3% incidence of spontaneous ventricular fibrillation in the control group of animals. However, when the posterior hypothalamus was stimulated via stereotaxically placed electrodes during coronary occlusion, the incidence of ventricular fibrillation increased tenfold to 63%. The demonstration that electrical stimulation of peripheral and central neural structures in anesthetized animals results in increased vulnerability to ventricular fibrillation does not constitute direct evidence that such sympathetic adrenergic mechanisms operate in the conscious animal.

Study of neural and psychologic mechanisms in the conscious animal poses many difficult methodologic problems. Induction of ventricular fibrillation and subsequent defibrillation does not permit the systematic analysis of the effects of neural and psychologic inputs on the heart. Defibrillation is traumatic and results in autonomic neural tone alterations and in changes in the psychologic state of the animal. To obviate these limitations, the repetitive extrasystole (RE) threshold is utilized as the marker for vulnerability to ventricular fibrillation.[19] The repetitive extrasystole is an electrophysiologic event which reliably tracks the threshold for ventricular fibrillation and has the advantage of not being perceived by the animal. Using this method of study, substantial increases in vulnerability to ventricular fibrillation were demonstrated when dogs were stressed in an aversive environment.[20, 21, 22] The RE threshold in a tranquil environment was taken as the control value. When the animals were placed in a sling where they had previously been immobilized and been subjected to aversive shock conditioning, the threshold decreased by 40% or more although no further shocks were administered. This enhancement in vulnerability was accompanied by increases in heart rate, blood pressure and serum catecholamines.[23] Using the same experimental model, malignant ventricular arrhythmias were evoked repeatedly in dogs recovering from myocardial infarction when they were placed in the threatening sling environment.[24] Skinner and others[25] have demonstrated that pigs exhibit a higher incidence of ventricular fibrillation when coronary artery occlusion is performed in a stressful unfamiliar laboratory setting as compared to an environment to which the animals had previously adapted.

Stress-induced changes in vulnerability using the RE threshold method can be abrogated with tolamolol, a cardioselective beta-adrenergic blocking drug.[21] Khan et al.[26] have found a similar protective effect against ventricular fibrillation during coronary occlusion using propranolol. In Skinner's[25] experiments, however, propranolol did not protect against ventricular fibrillation, suggesting that such protection from beta-adrenergic blockade may be incomplete.

HUMAN STUDIES

In man, the neurohumoral changes and the cardiac responses to physical, thermal, environmental and emotional stresses have been extensively documented.[27, 28, 29, 30, 31, 32, 33] Taggart and coworkers[27, 28] have demonstrated increases in serum catecholamines and ventricular ectopic beat frequency during stresses induced by driving, car racing and public speaking. The evocation of memories of emotionally painful events from the past during clinical interviews resulted in the emergence of increased ventricular ectopic activity as demonstrated in studies by Sigler[29] and by Stevenson and coworkers.[30] Case reports by Wellens et al.[31] and Lown et al.[32] document the relevance of psychologic inputs in the provocation of ventricular fibrillation. In the former instance, an adolescent girl with the long Q-T syndrome developed repeated bouts of ventricular fibrillation provoked by frightening or loud sounds. Lown and his coworkers,[32] in a detailed study of a 39 year old man with a structurally normal heart, showed that psychologic factors played a major role in the inception of ventricular fibrillation.

In an attempt to study objectively the role of psychologic inputs on arrhythmia frequency, a structured 3-part psychologic stress test was employed with 19 patients, 8 of whom had been resuscitated from sudden death.[33] The test consisted of mental arithmetic, reading from Stroop color cards and a discussion of stressful life events, including the patient's current illness. A two-fold increase in ventricular premature beat frequency occurred during the test period as compared to the control period in the 19 patients studied. Malignant runs of ventricular tachycardia were provoked in one patient who had repeated cardiac arrests in the past. This arrhythmia subsided completely following reassurance alone. In these patients, the passive evocation of various autonomic reflexes, including the "dive" reflex, did not provoke significant arrhythmias, except in one patient. This patient was emotionally upset at the time the "dive" reflex was elicited. Thus, in the absence of inputs from higher neural centers, autonomic nervous system activation is unlikely to stimulate ventricular arrhythmias.

These clinical observations demonstrate that ventricular arrhythmia in man is an episodic occurrence which is sensitive to phasic neural inputs evoked by sympathetic arousal. Such arrhythmias in patients with ischemic heart disease are presumed to reflect electrical instability of the myocardium and predisposition to sudden death from ventricular fibrillation.[34]

ROLE OF THE VAGUS

A differing view, that death results from asystole rather than ventricular fibrillation, has been suggested by the work of Richter[35] in rats. Wolf,[36] enlarging on this observation, has suggested that cardiac slowing and asystole may result from a vagal mechanism, possibly related to the so-called "dive" reflex. This mechanism of death in man, however, is most unlikely as vagal slowing invariably results in the emergence of escape pacemakers which govern cardiac activity. Resuscitation studies by Liberthson and others[37] have shown that asystole and bradyarrhythmias accounted for less than 10% of sudden deaths, the remainder being due to ventricular fibrillation. The finding of asystole in an agonal electrocardiogram does not preclude ventricular fibrillation as the terminal mechanism which had further deteriorated to complete cardiac standstill. When asystole is the cause of death in man, it is not due to psychologic factors, but rather to extensive cardiac injury, severe hypoxia or electromechanical dissociation. In the setting of acute infarction, vagal overactivity may indeed result in slow heart rates, atrio-ventricular block and ventricular arrhythmias including ventricular fibrillation. However, in these instances, the infarct zone is usually small, located in the inferior wall of the heart, and attended by a good prognosis. It is relevant to note that vagal stimulation in anesthetized and conscious animals results in significant protection from ventricular fibrillation.[22, 38, 39] Such protection is thought to accrue not from the direct effects of the vagus, but from reflex inhibition of sympathetic outflow to the heart.[38] Reduction of tonic sympathetic discharge to the heart also exerts beneficial effects on ventricular arrhythmias in man. In the clinical setting, use of cholinergic agents such as edrophonium results in suppression of ventricular premature beats and even termination of ventricular tachycardia.[40]

REDUCTION IN SYMPATHETIC TONE

Sleep may reduce both the frequency and grade of ventricular premature beats.[41] This salutary effect has been ascribed to a reduction of sympathetic tone and a preponderence of vagal tone during sleep. The effects of an altered state of consciousness produced by a non-cultic form of meditation have also been examined.[42, 43] This method of relaxation presumably induces a decrease in central sympathetic outflow and thereby promotes reduction in ventricular premature beat frequency. Learned suppression of ventricular premature beats due to an increase in heart rate is presumably due to overdrive suppression of an ectopic ventricular focus.[44, 45] Beta-adrenergic blockade with pharmacologic agents may also be expected to decrease ventricular arrhythmias because of lessened sympathetic neural drive on the heart. Large epidemiologic studies[46, 47] have shown that the incidence of sudden death can indeed be reduced by beta-adrenergic blocking drugs. The failure of such blockade to afford complete protection from sudden death implies the operation of additional mechanisms, either neural or cardiac, involved in the genesis of ventricular fibrillation. More recently, Rabinowitz and Lown[48] have shown that administration of serotonin precursors protects the heart from vulnerability to ventricular fibrillation in anesthetized dogs. The mechanism of action is presumably mediated by a reduction in sympathetic outflow to the heart secondary to increased central serotonin accumulation.

CONCLUSION

The experimental and clinical evidence presented in this brief review indicates that the CNS has a modulating effect on electrical stability of the heart and provides a source of risk for ventricular fibrillation in the electrically unstable heart. Central sympathetic activation leads to a neurohumoral response resulting in release of catecholamines and increases in heart rate and blood pressure. These changes destabilize the heart electrically, permitting the emergence of ventricular fibrillation. The reduction in tonic and phasic CNS sympathetic activity may reduce susceptibility to this arrhythmia. The delineation of precise neurophysiologic and biochemical mechanisms through which the CNS acts awaits further clarification. The weight of present evidence, however, permits the conclusion that psychophysiologic events contribute to the provocation of ventricular fibrillation and sudden death. ☐

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LETTER TO THE EDITOR

To The Editor:

As with many other State agencies, the S. C. Department of Health and Environmental Control is confronted with increased demands for services and a concomitant reduction in state and federal appropriations. The Bureau of Laboratories is faced this fiscal year with the decision either to reduce services or charge fees for additional laboratory examinations.

On September 11, 1979, the Board of Health and Environmental Control approved charging a fee of $2.00 for each culture for *Neisseria gonorrhoeae*. This fee will be imposed beginning November 1, 1979. Billing will be monthly by computer, and included with current laboratory test charges.

The fees for tests performed in the Bureau of Laboratories reflect the cost for performing the examination, which include all technical and administrative costs. The usual excellent quality of the finest medical laboratory in South Carolina will prevail.

Sincerely,
Arthur F. DiSalvo, M.D., Chief
Bureau of Laboratories
S. C. Department of Health and
Environmental Control
2600 Bull Street
Columbia, S. C. 29201

Quinamm™

AVAILABLE ONLY ON PRESCRIPTION

Brief Summary

INDICATIONS: For the prevention and treatment of nocturnal recumbency leg muscle cramps, including those associated with arthritis, diabetes, varicose veins, thrombophlebitis, arteriosclerosis, and static foot deformities.

CONTRAINDICATIONS: Because of the quinine content, Quinamm is contraindicated in women of childbearing potential, in pregnancy, in patients with known quinine sensitivity, and in patients with glucose-6-phosphate dehydrogenase deficiency. Hemolysis (with the potential for hemolytic anemia) has been associated with a G-6-PD deficiency in patients taking quinine.

PRECAUTIONS: Thrombocytopenic purpura may follow the administration of quinine in highly sensitive patients. Recovery will follow withdrawal of the medication.
Cinchona alkaloids, including quinine, have the potential to depress the hepatic enzyme system that synthesizes the vitamin K-dependent factors. The resulting hypoprothrombinemic effect may enhance the action of warfarin and other oral anticoagulants.

ADVERSE REACTIONS: Aminophylline may produce intestinal cramps in some instances, and quinine may produce symptoms of cinchonism, such as tinnitus, dizziness, and gastrointestinal disturbance. If ringing in the ears, deafness, skin rash, or visual disturbances occur, the drug should be discontinued.

DOSAGE AND ADMINISTRATION:
1 tablet upon retiring. When necessary, 1 additional tablet may be taken following the evening meal.

Product Information as of September, 1977

U.S. Patent 2,985,558

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8-3305 (Y371A)

PRELIMINARY OBSERVATIONS ON A NEW NON-DRUG METHOD FOR CONTROL OF HYPERTENSION*

ELMER E. GREEN, Ph.D.
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In 1974 a medical writer for a Kansas City newspaper visited our laboratory to get information on biofeedback as a medical tool. In order to demonstrate that biofeedback machines do nothing but feed back information about on-going physiological processes, we gave her a thirty-minute training session with a portable temperature machine, using autogenic phrases we had adapted from Schultz and Luthe[1, 2] for relaxing the body and increasing the temperature of the hands and let her take the machine home "to experiment with." A week later she returned the machine and said, "You may be interested to know that I got rid of my hypertension last week."

Pressed for an explanation, she said that she felt that stress was her real problem, and that her blood pressure, which had been averaging about 150/90mm Hg (while taking Valium for anxiety tension reduction) would decrease if she could relax. In addition, she said, "Why shouldn't it decrease; hand warming means blood-flow control, and after all, blood is blood." By an ingenious non-sequitur, she had provided herself with a rationale for attempting self regulation of hypertension. She also said that she stopped taking her medication after her visit with us and had made an appointment with her cardiologist to get a blood pressure check at the end of the week — and it was normal when taken in the doctor's office. He suggested that she continue without the Valium and check with him again.

She was asked to get her doctor's permission before continuing the practice of temperature control as it could have been a labile reaction that might just as quickly reverse. The incident was thought-provoking, though, and later it was mentioned as an unexplained event to a group of physicians attending a biofeedback workshop. One of them, a surgeon, made the semi-serious comment, "Perhaps she gave herself a reversible sympathectomy." In explanation, he said that before the appearance of modern drugs for controlling hypertension, he had often (in serious cases) cut the sympathetic nerves leading to the legs of hypertensive patients in order to induce vasodilation which caused a significant lowering of blood pressure. Benefits were not permanent and there were certain unpleasant side effects, but the procedure was often useful. The surgeon wondered if when she practiced hand warming, using the general whole-body autogenic phrases (progressively relaxing from the feet upward and warming her hands), might she have significantly warmed her feet — as in a sympathectomy?

Later, when a physician in Topeka asked if we could help a patient with hypertension not satisfactorily controlled by medication (her blood pressure was averaging about 190/110mm Hg), we agreed to try. This patient ("AD" of Table I) was a 38-year-old woman who had been on hypertension medication for sixteen years.[3] She was taking four Aldomet and four Inderol tablets daily, as well as medications for control of a kidney infection. Results were highly encouraging. In five months, her blood pressure was normal and her physicians had gradually reduced to zero her kidney and hypertension medications. Results from eleven additional patients are discussed below. but first it may be useful to outline the rationale[4] of biofeedback-aided self regulation training.

RATIONALE

Electrophysiological studies indicate that every perception of outside-the-skin (OUTS) events (see upper left box in Figure 1) has associated with it (Arrow 1) electrical activity in

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* From the Menninger Foundation, Topeka, Kansas 66601. The authors acknowledge the assistance of Michael Prohaska and Shirley Anderson in preparing this report, and of the following psychophysiological therapists whose patients are included in this on-going study: Steven Fahrion, E. Dale Walters, Verlyn Norris, Richard Gloor, William Hale, and Jeffrey Nichols.

TABLE I: BIODATA AND TRAINING RESULTS WITH TWELVE HYPERTENSIVE PATIENTS

BIODATA \ PATIENTS	AD	ZL	AQ	CF	MJ	GR	FG	FCG	AA	NS	BS	RZ
SEX, AGE AT INTAKE	F 38	M 57	F 44	M 28	F 40	M 57	F 27	M 39	F 53	F 52	F 38	F 56
DATE OF INTAKE	Feb 75	May 75	May 77	Jul 77	Nov 77	Dec 77	Feb 78	Apr 78	May 78	May 78	May 78	May 78
YEARS OF DIAGNOSED HYPERTENSION	16	39	3	3	2	30	1	15	10	1	3	3
YEARS OF ANTIHYPERTENSIVE MEDICATION	16	16	0	3	0	10	1	3	0	0	3	0
STARTING MEDICATION INDEX (MI)	38	25	0	58	0	50	25	19	0	0	50	0
MI AT FOLLOWUP	0	17	0	0	0	0	0	0	0	0	0	0
AVG. BLOOD PRESSURE IN FIRST 10 SESSIONS	169/110	133/95	139/86	130/83	143/94	142/88	138/89	133/92	142/82	144/99	136/88	149/105
AVG. BLOOD PRESSURE IN LAST 10 SESSIONS	145/92	130/80	118/83	128/80	145/90	123/81	141/91	131/85	141/79	N/A	125/79	146/102
CHANGE IN BLOOD PRESSURE	-24/-18	- 3/-15	-21/- 3	- 2/- 3	+ 2/- 4	-19/- 7	+ 3/- 2	- 2/- 7	- 1/- 3	N/A	- 9/- 9	- 3/- 3
AVG. BLOOD PRESSURE AT FOLLOWUP	132/84	On Going	120/80	137/84	142/94	135/83	133/84	130/85	135/85	On Going	130/80	On Going
WEEKS TO TERMINATION OF DRUGS	25	On Going	N/A	19	N/A	71	1	21	N/A	On Going	32	On Going
WEEKS TO TERMINATION OF TRAINING	26	On Going	16	23	13	54	21*	28	33	On Going	34	On Going
WEEKS TO FOLLOWUP AFTER TRAINING	104	On Going	52	39	31	17	22	5	11	On Going	10	On Going

*UNABLE TO CONTINUE TRAINING

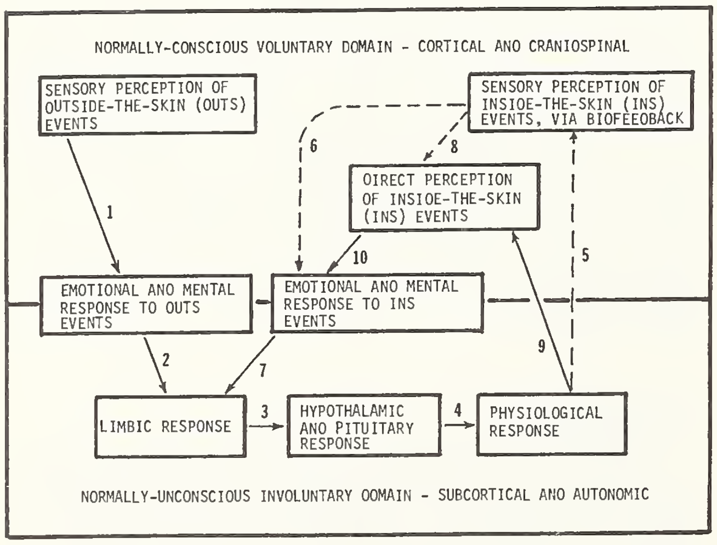
NORMALLY-CONSCIOUS VOLUNTARY DOMAIN - CORTICAL AND CRANIOSPINAL
SENSORY PERCEPTION OF OUTSIDE-THE-SKIN (OUTS) EVENTS
SENSORY PERCEPTION OF INSIDE-THE-SKIN (INS) EVENTS, VIA BIOFEEDBACK
DIRECT PERCEPTION OF INSIDE-THE-SKIN (INS) EVENTS
EMOTIONAL AND MENTAL RESPONSE TO OUTS EVENTS
EMOTIONAL AND MENTAL RESPONSE TO INS EVENTS
LIMBIC RESPONSE
HYPOTHALAMIC AND PITUITARY RESPONSE
PHYSIOLOGICAL RESPONSE
1
2
3
4
5
6
7
8
9
10
NORMALLY-UNCONSCIOUS INVOLUNTARY DOMAIN - SUBCORTICAL AND AUTONOMIC

FIGURE 1. Simplified operational diagram of "self-regulation" of psychophysiological events and processes. Sensory perception of OUTS events, stressful or otherwise (upper left box), leads to a physiological response along Arrows 1 to 4. If the psychological response is "picked up" and fed back (Arrow 5) to a person who attempts to control the "behavior" of the feedback device, then Arrows 6 and 7 come into being, resulting in a "new" limbic response. This response in turn makes a change in "signals" transmitted along Arrows 3 and 4, modifying the original physiological response. A cybernetic loop is thus completed and the dynamic equilibrium (homeostasis) of the system can be brought under voluntary control. Biofeedback practice, acting in the opposite way to drugs, increases a person's sensitivity to INS events and Arrow 8 develops, followed by the development of Arrows 9 and 10. External feedback is eventually unnecessary because direct perception of INS events becomes adequate for maintaining self-regulation skills. Physiological self-control through classical yoga develops along the route of Arrows 7-3-4-9-10-7, but for control of specific physiological and psychosomatic problems biofeedback training seems more efficient.

both conscious and unconscious CNS structures, those involved in emotional and mental responses.[5] The two boxes labeled "emotional and mental responses . . ." have been placed at the middle of the diagram, divided by the horizontal center-line into conscious and unconscious parts, in order to indicate their two-part nature. The next box, called 'limbic response," is placed entirely in the unconscious section of the diagram, though some neural pathways lead from limbic structures directly to cortical regions, implying that "information" from limbic processes can reach consciousness.

The limbic system has been intensely studied in both animals and humans in the years since Papez' historic paper[6] outlined the possible functions of the limbic system in emotional responses. MacLean coined the phrase "visceral brain" for the limbic system[7] and others have referred to it as the emotional brain, but the important point is that emotional states and visceral processes are correlated with electrophysiological activity in the limbic system. Of major significance is the fact that the limbic system is connected by many pathways, represented by Arrow 3, to the central "control panel" of the brain, the hypothalamus. Though the hypothalamus weighs only about four grams, it regulates a large part of the body's automatic neural machinery and, in addition, it controls the pituitary gland. The pituitary, the so-called "king gland" of the body, is at the top of the hormonal hierarchy and its action precipitates or triggers changes in the homeostatic levels of other glandular structures. In other words, limbic signals are essentially converted to autonomic and hormonal processes through the transducing machinery of the hypothalamus and pituitary.

The hypothalamus and pituitary maintains physiological balance in response to variations in internal signals fed back from tissues and organs, but they do not initiate change as far as we know. Instead, changes in physiological balance seem to proceed for the limbic level of the neurological control hierarchy, as implied for example in Masserman's work with sham rage in cats.[8] In animals the limbic system responds to *perception*, as shown by several dozen ethological studies, and in humans the limbic system responds to both perception and *imagination*. In some people, imagination is often more powerful that perception, and they are more physically upset by what they imagine than by what they actually perceive.

With these psychoneurological concepts in mind it is not difficult to understand how news from a telephone message can result in a surge of high blood pressure. The perception of OUTS events leads to limbic-hypothalamic-glandular responses, and, of course, physiological change is the inevitable consequence. Both genetic structure and previous conditioning are factors in determining the specific nature of a physiological response, but neither operates in a vacuum, independent of the visualization factor.

Almost all of this was known by 1958[5], but what was not known was the fact that if we can detect what is going on inside our skin we can often voluntarily change it in a direction we choose. If a physiological change from the box at the lower right in the diagram is "picked up" by a sensitive electrical transducer (Arrow 5) and displayed to the person with a meter, or made audible by a tone so as to feed back continuous physiological information, then there ensues (Arrow 6) a "new" mental-emotional response, a new response to normally-unconscious inside-the-skin (INS) information. The new mental-emotional response, is associated with an appropriate limbic response (Arrow 7) that combines with, or replaces, or modifies, the original response (Arrow 2), and in turn changes the pattern of hypothalamic firing and pituitary behavior. This alters the physiological state and it is this physiological change that is signaled by the feedback monitor to the conscious cortex.

A biocybernetic control loop is thus established as a result of providing the conscious cortex with information about normally-unconscious INS processes. Closing the biocybernetic control loop literally means bridging the normal gap between conscious and unconscious processes, voluntary and involuntary. It is noteworthy that we often cannot describe in words what we do existentially to control a normally unconsicous process, we do not become directly aware of the neural pathways and muscle fibers involved any more than we become aware of which cortical and subcortical nerves are involved in playing tennis. But, as in tennis, when we get external objective feedback we can learn to modify the internal "set up" so as to bring about change in the desired direction.

When a person watches a temperature feedback meter, it is not the movement of the needle that brings about self-regulation of blood flow, rather it is (1) the imagining and visualization of what is wanted (e.g., increasing warmth in the hand, or decreasing heart rate) and (2) the information of "what works," as revealed by the movement of the needle.

We have found it helpful with new patients to explain the rationale in order to dispel mysteries and to foster a mental set within which physiological self-regulation is relatively easy to learn. In terms of a popular metaphor, it is as if the left cortex must be persuaded of, or at least tolerate the possibility of, the rational nature of neurological self-regulation, before it can allow the right cortex to construct a psychophysiological visualization (pictorial and/or kinesthetic) to bring about the regulation. As far as we know, this has not been specifically demonstrated through research, but we have noticed that our most labile hypertensive patients are not primarily discursive, deductive thinkers (said to be a left cortex attribute), rather they are intuitive and tend to perceive or misperceive in gestalt-like ways (said to be a right cortex attribute).

Professional people and scholars who become our patients often find the rationale diagram of particular value, and remark that they are "beginning to understand" how the entire process works, and are beginning to *believe* (correlated with a limbic process, in our view) that maybe they can make it work for them. This is in contrast to Johannes Schultz' observation in Autogenic Training that pedants and other scholarly types were the ones most difficult to teach. They could think of many reasons why Autogenic Training could not work, and their consequent attitude and negative visualization (again correlated with a limbic response) interfered with learning.

If, however, a sophisticated patient says that the rationale may only strengthen a placebo effect, we point out that it is an error not to recognize that the placebo effect is merely a subdivision of the general "self-regulation effect." When visualization and expectancy are activated through the use of a placebo, the effect almost invariably fades as time passes because it is attached to an inactive medical factor coupled with fading visualization and expectancy. But when visualization and expectancy are attached to one's own volitional effort, then a true skill can be developed, embodying genuine self regulation of the autonomic nervous system, and it does not fade away any more than knowing how to ride a bicycle fades away. For training to be most effective, the patient must thoroughly understand that self regulation is psychological, neurological, and practical.

TRAINING PROCEDURE

(1) At the first session, after physician referrals and entrance requirements are met, it is explained to the patient that the essence of self-regulation of any kind is proper visualization. This is true whether the person is an Olympic athlete trying to break the high jump record or a patient with Raynaud's disease trying to control blood flow in the hands. In both cases a trial and error method is used. It is explained that athletes use feedback (information about trials) in order to perfect their skills and no skill was ever learned without feedback. Guided missiles, computers, and other devices programmed for self-regulation all use performance information that is fed back in order to make corrections in behavior. And the same is true of biofeedback trainees — except that they generate their own programs and strategies for self regulations with the aid of a teacher, the psychophysiological therapist.

(2) Next, a neurological rationale of self regulation is provided, explaining that for self regulation, appropriate information must reach the cortex. If the patient is a neurologist, the explanation is considerably more technical than for a high school student, but in either case the rationale diagram of Figure 1 is discussed.

If the rationale seems a burden to the patient, we conclude by saying that none of the foregoing is of great importance for developing skill in self-regulation, any more than it is necessary to know neuroanatomy in order to play golf. Nevertheless, it is useful to know that all of the neurological machinery is neatly organized and there are no mysteries about the major functional processes. They can be confident that if someone says, "That's crazy, no one can recover from hypertension that way," it is merely ignorance speaking, no matter how neurologically educated the critic. Neurological sophistication and psychoneurological sophistication are not necessarily the same thing.

(3) The discussion of the rationale is followed by a determination of the patient's own treatment goals. If self-regulation training is to fully succeed, it is necessary for the patient to assume self-responsibility, and his or her own definition

of treatment goals is one of the important steps in the process.

(4) Then the first temperature training session is given. We attach a thermister to a finger of the patient's dominant hand and explain that the temperature of the hand is dependent on smooth muscle tension in blood vessel walls, which in turn depends on the firing rate of sympathetic nerves which is regulated by the hypothalamus. In other words, in learning to warm the hands, the patient is actually learning to regulate the sympathetic outflow to the periphery.

(5) We then conduct a fifteen minute autogenic session with heaviness, warmth, and inner quietness phrases while we observe and record the temperature changes. We teach the patient how to use the feedback machine and assign it for home practice. We also ask patients to purchase a sphygmomanometer and instruct them in the use of it.

(6) All trainees are asked to practice twice daily at home, using autogenic phrases in conjunction with the temperature machine. They are also asked to fill out a questionnaire following one session each day — recording temperatures and blood pressures (before and after the session) and reporting their bodily sensations, emotions, and thoughts. At first, patients focus on hand warming and when that is learned, they begin foot warming.

(7) After the first week or two of home training, it is suggested that trainees begin to discontinue the use of autogenic phrases and to replace them with their own personal visualizations relating to temperature control. (Patients may imagine such things as lying on a warm beach.) Finally the control of temperature can be shortened to "turning on" a kinesthetic sensation which brings about vascular relaxation in a minute or two. Above all, we do not want patients to become bound by the idea that the use of phrases or other procedures, such as listening to music or body-control suggestions on cassette tapes, are necessary in order to initiate a desired kind of physiological behavior.

(8) Within the first week or two, we teach the patient a simple breathing exercise which we call triangular breathing. The breathing pattern consists of deep, but not forced, inhalations and exhalations at a constant rate of flow, with no pauses at tops or bottoms of cycles. When wired to a respiration gauge, the patient makes a triangular record on a polygraph stripchart. At first each inhalation-exhalation cycle takes about eight seconds, but as patients learn to breathe more quietly and deeply, the rate often slows to a more comfortable ten or twelve seconds/cycle.

(9) Most patients come to the Biofeedback and Psychophysiology Center for training once or twice a week. The accumulated home temperature and blood pressure reports are discussed with the psychophysiological therapist at these sessions.

(10) At intake, we send the patient's referring doctor a note describing the self-regulation training procedures to be used and every eight weeks thereafter we send a progress report. When the therapist and patient feel that gains have been maximized, training is terminated and a final report summarizing what was accomplished is sent to the patient's physician.

MEDICATION INDEX

The drugs most frequently used for direct hypertension control are categorized as diuretics, central nervous system controllers, and peripheral nervous system controllers. Whatever the general category, we have found it useful to derive a medication ratio for each drug consumed, by dividing the amount taken daily by the "norm" for that drug. Physicians differ in their use of specific medications, so for purposes of standardization, our norms are derived from the Physicians Desk Reference (PDR). The norm is defined as the dosage that is one-third of the way from the smallest recommended daily therapeutic dosage to the largest recommended therapeutic dosage. For example, if the PDR's minimum and maximum daily therapeutic dosages are 50 and 100 mg, as with hydrochlorothiazide, then the norm is 67 mg.

The diuretic ratio, the central ratio, and the peripheral ratio are then added together and multiplied by 33 to obtain (in arbitrary units) the Medication Index (MI), as shown in Figure 2. Note that the three categories of drugs are weighted equally (33) in this formula. In the absence of accepted research-based findings, it is useful to avoid assumptions about central versus peripheral versus diuretic effectiveness. In any event, the main value of the index is not in making comparisons from patient to patient, but in charting the course of drug consumption in an individual.

The names of the drugs shown in Figure 2 are generic rather than commercial. If a particular

FIGURE 2. DEFINITION OF A MEDICATION INDEX FOR HYPERTENSION PATIENTS, AND MEDICATION NORMS FOR EIGHT OFTEN-USED DRUGS.

DIURETICS (D)	DAILY THERAPEUTIC DOSAGE RANGE*
1. HYDROCHLOROTHIAZIDE	50 to 100 mg. NORM 65
2. TRIAMTERENE	100 to 200 mg. NORM 135
3. CHLORTHALIDONE	50 to 100 mg. NORM 65
CENTRALLY ACTIVE (C)	
1. CLONIDINE HYDROCHLORIDE	0.2 to 0.8 mg. NORM 0.4
2. PROPRANALOL HYDROCHLORIDE	160 to 480 mg. NORM 280
3. METHYLDOPA	500 to 2000 mg. NORM 1000
PERIPHERALLY ACTIVE (P)	
1. HYDRALAZINE HYDROCHLORIDE	100 to 200 mg. NORM 133
2. GUANETHIDINE MONOSULFATE	25 to 75 mg. NORM 42

$$MI = \left(\frac{D\ DOSAGE}{D\ NORM} + \frac{C\ DOSAGE}{C\ NORM} + \frac{P\ DOSAGE}{P\ NORM}\right) \times 33$$

*From 32nd Edition (1978) of Physicians Desk Reference.

commercial preparation is composed of drugs of two different categories, then the amounts in each category are treated independently, and two ratios are calculated. On occasion a patient takes two kinds of drugs in a single category. In such a case, two ratios are computed and added together to give the total ratio of that category in the overall index.

A Medication Index is a necessary tool in evaluating a patient's progress. We mention this because one physician said, "Some of your patients didn't have pressures above 140/90mm Hg when they started training, so even though they were able to eliminate medication you did not demonstrate that they were able to control blood pressure." Technically this comment is correct, but the fact that the MI went to zero while blood pressure remained constant is of importance. Otherwise the medication was not needed. More importantly, side effects are abolished and medical costs are reduced. If hypertension control costs patients $1000/year, then twenty million patients in one decade will spend 200 billion dollars.

Whatever the answer in any particular case, it is clear that a training program in which medication can be reduced while the blood pressure is at least not allowed to rise, is a step in a healthy direction, both physical and financial.

OBSERVATIONS AND DISCUSSION

Data on our first twelve patients who were given psychophysiological training for hypertension control, including vasodilation in the legs, are summarized in Table I. Blood pressure data

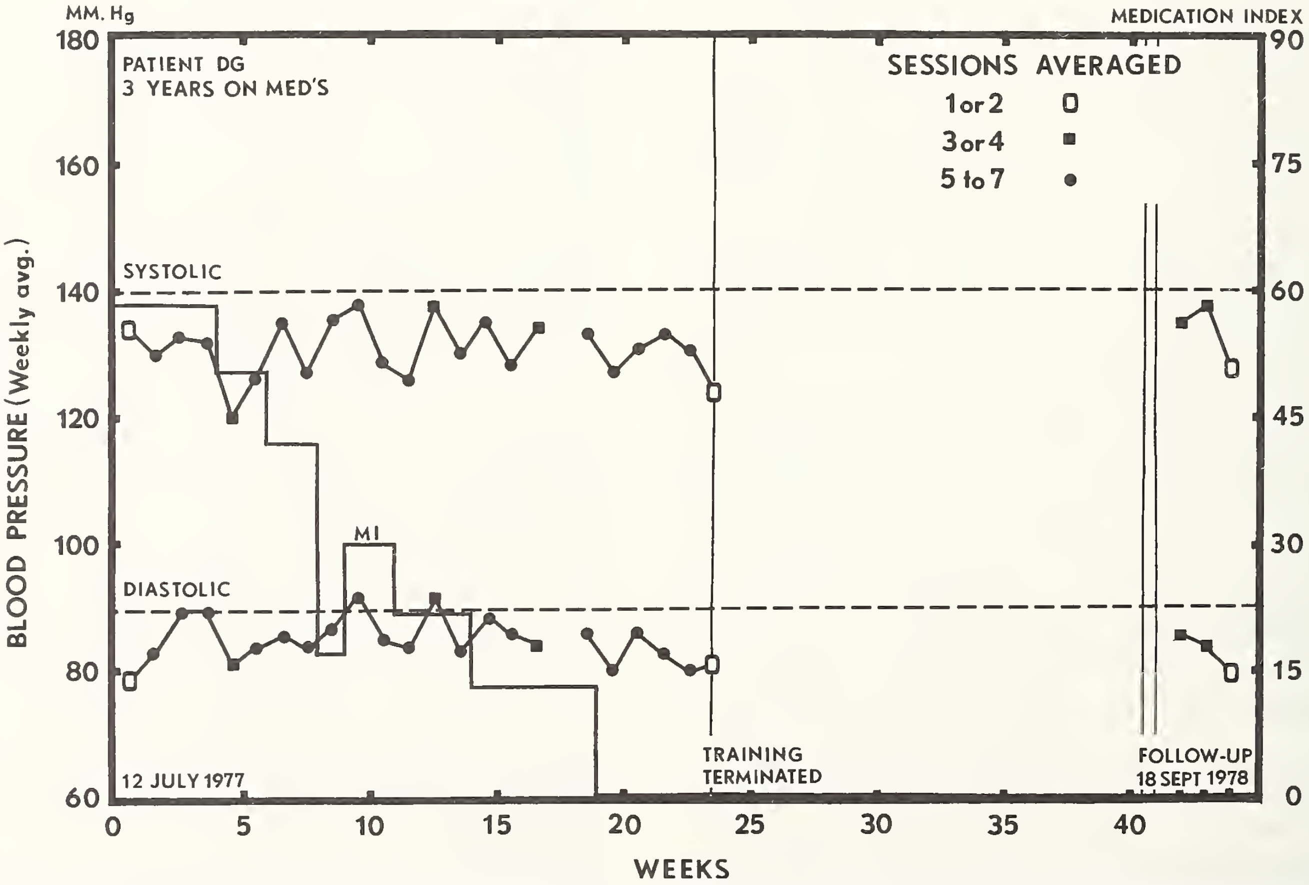

FIGURE 3. Graph of blood pressure and Medication Index of a 28-year old man during psychophysiological therapy for elimination of hypertensive medications.

and the MI are also plotted for each patient, as illustrated by the graph for patient CF (Figure 3). In Table I, the subject is identified as a male, age 28 at intake. Hypertension had been diagnosed three years earlier and at that time he was put on medication. He was a business man who resented the side effects of the medications and hoped that he could reduce or eliminate them while keeping his blood pressure normal. He was taken completely off medication by his cardiologist after 19 weeks and terminated training after 23 weeks with normal blood pressure. Thirty-nine weeks after termination of training his readings were still normal when measured several times over a period of three weeks.

The "Average Blood Pressure in First Ten Sessions" of Table I refers to home-training results and is invariably less than the pressures reported by the referring physician. In the case of GR, for instance, the physician reported pressures ranging from 196/120 to 150/95mm Hg, but no systematic records were available and that information was not included in the Table. In fact, systematic pre-training records were available on only two subjects. Our present procedure, however, includes a pre-training baseline phase of recording pressures at home.

Since systematic pre-training baselines were not obtained with this first group of patients, the average pressures in the first ten home-training sessions are extremely conservative estimates of the patients' average hypertensive states at intake. The average is conservative because pressures in several patients dropped rapidly in the first ten sessions. Although conservative, the average pressure of the first ten training sessions is a useful indicator against which to compare the average of the final ten sessions.

Of much greater importance in this particular compilation of observations is the change in Medication Index between "starting" and "followup." In six of seven cases in which medications were used for hypertension control, the MI was reduced to zero. In the other patient, ZL, the MI was reduced from 25 to 17. Since his training is on-going we cannot yet give a final figure. In addition to the changes in MI, the blood pressure readings were also appreciably lowered in five of these seven patients. Data from the other two patients, FÇG and FG, showed little change.

Of the twelve patients, five were not taking medication and two of these patients (AQ and AA) practiced the exercises regularly. AQ's pressures dropped considerably, but AA's averages did not change. The other three patients in the non-medication group did not practice and achieved no noticeable pressure drops.

Interestingly, the patients who had a high Medication Index when they came into the training program were the ones who seemed to do the best. Those who were not taking drugs were often the poor practicers and sometimes admitted they were poor candidates for self-regulation therapy.

In illustration of the need for motivation, one of the most interesting subjects was RZ. She was not on medication and her pressures varied from 170/120 to 140/95mm Hg, with an average of about 150/105mm Hg when she began training. In five weeks, she practiced only five times and bragged about the fact that she was a very poor patient. After 20 weeks, during which time she rarely practiced, it was mentioned to her that in a few years she would be at retirement age, but that from our knowledge of actuarial tables we estimated her life expectancy to be about sixty-five years and one month. It was too bad that after a lifetime of hard work she would be able to draw social security benefits for only about one month. That day she began practicing earnestly and her first trial was 136/88mm Hg. She later joined a training group with six other patients.

An interesting phenomenon noticed in working with hypertensive patients is the change in lifestyle in successful cases. For instance, patient GR (a psychiatrist) says he now looks forward to pausing at stop lights so that he can practice his relaxation exercises. This is in direct contrast to his former behavior, hurrying to get his car through every intersection. Incidentally, using stop lights as "relaxation signals" has been one of our most effective ways of reminding people that relaxing when appropriate must become a way of life.

GR also reported that for the first time in many years he began to notice the trees, the flowers and gardens, the sky, and that his feeling was entirely different about himself. He said, "I'm becoming more childlike, not childish mind you, but childlike. I see things I never saw before and am taking a new delight in life." Statements of this kind have become almost routine with patients as blood pressure and medication levels are successfully reduced. There is nothing surprising about this, of course, since hypertension in a specific patient may be related to stress more than to any other factor in his life. After people learn to "unstress" at will, they naturally have a different feeling about life, and generally become more efficient at whatever they do.

It is worth noting that all Olympic athletes get to the top by "learning to relax at every stroke." Relaxation after tension is necessary for success in any activity. If an athlete becomes so tense that the relaxation phase is destroyed, he or she may say they were "psyched out." The difference between the top performers and those who never quite make it includes, as one important factor, the ability to turn off the effects of stress at will.

It is too early to say what the limits of self regulation training may be, but from the general results already reported to the Biofeedback Society of America,[10] it is clear that we can not at this time, in good conscience, tell a patient what can *not* be done. □

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ABILITY OF BIOCHEMICAL AND PERSONALITY PARAMETERS IN DISCRIMINATING BETWEEN FITNESS GROUPS AND AGE LEVELS*

A. H. ISMAIL, H.S.D.
R. J. YOUNG, Ph.D.

INTRODUCTION

A variety of physiological changes accompany the aging process and reduce the older individual's capacity for physical work. Collectively, they reflect a decline in physical working capacity[1] and include decreased maximal heart rate, decreased musclar strength, and increased blood pressure to mention but a few. Furthermore, improvement in physiological functioning has been demonstrated in middle-aged and older subjects as a result of participation in a regular exercise program.[1, 2, 3]

Recent studies indicate that physically trained and untrained individuals differ significantly on several personality characteristics, particularly those dealing with emotional stability, security, self-confidence, and anxiety.[4, 5, 6, 7, 8, 9, 10, 11] Differences have also been found between trained and untrained subjects in several biochemical compounds which are known to be related to specific behavioral patterns and, in most instances alterations in these compounds are observed as a result of participation in an exercise program.[11, 12, 13, 14]

Compounds such as corticosteroids, catecholamines, glucose, cholesterol, and testosterone have been found to be related to several affective conditions.[13, 15, 16, 17, 18, 19] For example, competitiveness and aggression,[16] extraversion and feelings of depression and fear[18] have been shown to be associated with elevated cholesterol levels. Catecholamines have been observed to be liberated in a variety of stress states[12, 20, 21] and to reflect the intensity of emotional reactions[22, 23]; and testosterone has been linked to aggression[24, 25, 26] and social behavior.[27] Most of these studies, however, used as subjects mental patients with extreme manifestations of psychosis,[26] depression, schizophrenia,[28] and anxiety[23] rather than members of the "normal" population.

In the present study, we have investigated the ability of selected personality and biochemical parameters in discriminating between two fitness groups (high and low fitness conditions) and two age levels (young and old) before and after a four month physical conditioning program.

METHODS

Subjects

The subjects were 58 male Purdue University faculty and staff members and local businessmen, 21 to 61 years, who volunteered to participate in a four month physical fitness program. Each participant was required to pass a physical examination by his family physician before being accepted into the program. Two age groups were established and designated as young (n = 14) and old (n = 14). The subjects in the two groups were matched on physical fitness condition using the fitness criterion of Ismail, et al.[29] Similarly, from the 58 volunteers, two fitness groups were established and designated as high-fit (n = 14) and low-fit (n = 14). The subjects in the two fitness groups were matched for age. (Tables 1 and 5).

Physical Fitness Program

The fitness program consisted of jogging, calisthenics and recreational activities. It was conducted three days per week between 12:00 and 13:30 hr. The intensity of exercise and the rate of progression were carefully designed to match the subjects' capabilities.

Physical Fitness Assessment

The protocol for assessing physical fitness consisted of a 10 minute bed rest at the end of which heart rate and blood pressure were recorded using standard clinical procedures. The subject then walked on a treadmill for 10 minutes at 3 mph with the grade being increased by 2 degrees every 2 minutes to a maximum of 8 degrees. Submaximal data were obtained during the last 30 seconds of the walk. Without stopping, the subject commenced running at a speed of 6 mph with the grade being increased each 2 minutes from 0 to a maximum of 10 degrees if exercise was not terminated beforehand. Heart rate was continuously monitored using an electrocardiograph and the subject indicated when he wanted to terminate the run, whereupon expired air and heart rate data were collected. Oxygen uptake (VO_2) was determined from expired air using Beckman O_2 and CO_2 analyzers. The percentage of O_2 and CO_2 obtained were applied to a nomogram[30] to obtain the true O_2 percentage. Oxygen consumption in liters was calculated using the true O_2 percentage and the expired air volume corrected to STPD. This volume was converted into liters per minute and divided by kilograms of body weight to obtain the desired unit of measurement. The same measures were obtained at the posttest as were collected at the pretest. Percentage lean body weight was estimated using the method of Wilmore & Behnke.[31]

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Physical fitness scores, based on the regression equation of Ismail, Falls & MacLeod[29] were obtained for each subject before and after the program. The regression weights and constant are as follows:

−1.33	(submaximal exercise pulse rate)
4.88	(percent lean body weight)
2.50	(maximal oxygen uptake ml/kg lean body weight)
−119.02	(submaximal minute volume ventilation/kg body weight)
−1.36	(resting diastolic blood pressure)
1.31	(resting pulse pressure)
61.90	(constant)

Variables

Pre- and post- program data were collected on the following variables:

Biochemical: Four biochemical compounds were selected because of their previously documented relationship to age and certain personality characteristics.[10, 15, 16, 17, 18] Serum glucose and cholesterol levels were measured by the Hycel Method, Hycel, Inc., Houston, Texas. Serum testosterone concentration was determined by an adaptation of the radioimmunoassay technique of Chen, et al.[32] using reagents supplied by Wien Laboratories, Inc., Succasunna, New Jersey. Free catecholamines were determined from a 24 hr. urine sample using ion-exchange resin columns supplied by Bio-Rad Laboratories, Richmond, California.

Venous blood samples were drawn from the antecubital vein, following a 10 minute bed rest, using a standard plain Vacutainer system. Two 10 cc Vacutainers of blood were extracted and allowed to stand for 15 minutes after which they were centrifuged at 3,000 rpm for 10 minutes and the serum was separated. All samples were obtained between the hours of 07:00 and 10:00, stored at −20°C, and assayed within two months. For consistency, the acidified 24 hr. urine sample was collected on a Sunday. A 50 ml aliquot was stored at −20°C and analyzed within two months.

Personality: Data were collected using the Cattell Sixteen Personality Factor Questionnaire — Form A,[33] the Eysenck Personality Inventory — Form A,[34] and the "In General" from of the Anxiety Scale of the Multiple Affect Adjective Check List.[35]

Statistical Analysis

The t-test was used to compare the young and old groups, and the high and low fit groups, before and after the exercise program.

At each test period several discriminant function analyses were perfromed and the three best personality variables were identified by the values of the discriminant function coefficients. These were combined with the biochemical compounds and final discriminant function analyses were performed on the pre- and post-program data for the age groups as well as the fitness groups. The discriminant function analysis allows interpretation based on the magnitude and direction of the coefficients obtained on each variable and reflects the relative importance of the variables in discriminating between the groups.

RESULTS

Age Groups: The means and standard errors of the selected physiological variables are presented in Table 1 to illustrate the differences between the two groups and the effects of the exercise program. From pre- to post-tests both the young and old groups significantly decreased in maximal HR ($p < .01$). The old group significantly increased in percent lean body weight (p

$< .05$), VO_2 max ($p < .05$), and physical fitness score ($p < .001$).

For interest, the pre- and post- program personality and biochemical data for the young and old groups are presented in Table 2. Only those personality variables used in the final discriminant function analyses are included. At the posttest, the young group scored significantly higher on Factor E (submissive vs. aggressive) ($p < .01$), lower on Factor M (practical vs. imaginative) ($p < .05$), and had a significantly ($p < .01$) lower urinary catecholamine excretion rate than the old group. From pre- to posttest, the young group significantly decreased in serum glucose level ($p < .05$) and the old group significantly decreased on Factor E ($p < .05$).

The pretest discriminant function coefficients for differentiating between the two age groups are presented in Table 3. The Mahalanobis D^2 of 1.5039, approximated by the F ratio of 1.16, was not significant. In contrast, the posttest Mahalanobis D^2 (Table 4) of 4.2655, approximated by the F ratio of 3.28, was significant at the .05 level. The magnitude of the coefficients indicated that Factors E (submissive vs. aggressive), G (expedient vs. conscientious), and M (practical vs. imaginative) are the best discriminators between groups. Surprisingly, the biochemical variables were not powerful discriminators as reflected by their coefficients. In general, the young subjects tended to be more aggressive, conscientious and practical and had lower catecholamine and glucose levels than the old subjects.

Fitness Groups: The means and standard errors of the selected physiological variables are presented in Table 5 to illustrate the differences between the two fitness groups and the effects of the exercise program. At the pre-test, the high-fit group was significantly lighter ($p < .01$), higher in percent lean weight ($p < .001$), lower in systolic ($p < .01$) and diastolic ($p < .01$) blood pressure, and higher in VO_2 max ($p < .001$) than the low-fit group. At the post-test, they were not significantly lighter ($p < .01$), higher in percent lean weight ($p < .001$), VO_2 max ($p < .01$) and physical fitness score ($p < .001$) than the low-fit group. From pre- to post-test, the high-fit group significantly increased in percent lean ($p < .05$) and reduced in maximal HR ($p < .01$). The low-fit group reduced significantly in systolic blood pressure ($p < .05$) and maximal HR ($p < .01$) and significantly increased in physical fitness score ($p < .01$).

The pre- and post- program biochemical and personality data for the high- and low- fit groups are presented in Table 6. Only those personality variables used in the final discriminant function analyses are included. At the pre-test, the high-fit group scored significantly higher on Factor M ($p < .01$) than the low-fit group. At the post-test, the high-fit group scored significantly lower on Factor L ($p < .01$) and higher on Factor M ($p < .05$) than the low-fit group. At the pre-test, the high-fit group had a significantly ($p < .05$) lower serum glucose level and a significantly ($p < .05$) higher serum testosterone level than the low-fit group. The low-fit group reduced significantly ($p < .05$) in serum glucose level from pre- to post-testing.

The pre-test discriminant function coefficients for differentiating between high and low fitness groups are presented in Table 7. The Mahalanobis D^2 of 4.5882, approximated by the F ratio of 3.53, was significant at the .05 level. The magnitude of the coefficients indicated that Factors M (practical vs. imaginative), H (shy vs. adventurous), and L (trustful vs. suspecting), were the best discriminators between the two groups. The biochemical variables were poor discriminators as reflected by their coefficients. In general, the highly fit subjects tended to be more imaginative, adventurous and trustful and had lower catecholamine, glucose and cholesterol levels and a higher testosterone level than their less fit counterparts.

The post-test (Table 8) Mahalanobis D^2 of 3.9219, approximated by the F ratio of 3.02, was also significant at the .05 level. The results were similar to those obtained at the pre- test. It is interesting to note that the catecholamine levels became the poorest discriminator among the seven variables which indicates some changes in the secretion rate by the low-fit group as reflected by the mean value during the posttest.

DISCUSSION

This study sought to determine the discrimination power of selected biochemical compounds and personality characteristics between two age levels and two physical fitness groups before and after an exercise program. Several studies have demonstrated that desirable physiological changes can be accrued as a result of participation in exercise programs.[1, 2, 3] Our results are in agreement with the findings obtained by those investigators.

The results obtained from the application of the discriminant function analysis involving the personality factors and the four biochemical compounds (when comparing the two age groups) tend to support the previous finding by Ismail and Young.[8] They have noted that the fitness program tends to stabilize the factors affecting personality as reflected by the reduction in the variability (a smaller standard error) found in post- test data. Therefore, the true difference between the two groups is more apparent at the post- program testing.

Factor E (submissive vs. aggressive) was found to be the most powerful discriminator between the two groups. Cattell[33] notes that this factor alters slightly with age and, in this study, slightly above average scores (indicating dominance and aggression) were obtained by the young group (Table 2). Moreover, the old group decreased significantly ($p < .05$) on this factor from pre- to post- testing, resulting in a significant ($p < .01$) difference between the groups at the posttest. Factor E is related to extraversion and Eysenck[34] has postulated that the extraversion scale of the EPI reflects the strength of the excitatory and inhibitory functions of the central nervous system. The difference between age groups, therefore, may reflect the effect of aging on the central nervous system.

Testosterone level has been related to aggression in both animals[24, 25, 36] and man[26, 37, 38] and the differential aggression levels observed in this study may be the result of hormonal changes associated with aging. The testosterone values obtained in this study are similar to reported averages of normal male subjects of comparable ages.[39, 40, 41] The old group had a slightly lower mean testosterone level than the young group at both the pre- and post- tests but it is interesting to note that the difference between the groups was not significant despite the twenty year age difference. Young, Ismail, Bradley, et al.[11] note that testosterone level is not purely a function of age, but is also dependent upon physical condition. In this study, no significant differences were found between the two groups in terms of physical fitness score.

Factor G, dealing with superego strength, was the second most powerful discriminator between the groups. Higher scores were observed in the young group indicating higher superego strength. Personality characteristics dealing with superego strength have been related to serum lipids.[8, 9, 28] Ismail & Young[13] found that hypercholesterolemia and hyperglycemia were related to low superego strength. Serum cholesterol is known to increase with age.[42, 43] In this study, higher cholesterol levels and lower superego strenth were observed in the old group.

At the posttest, the old group scored significantly ($p < .05$) higher on Factor M (practical vs. imaginative) than the young group. Individuals who score high on this factor possess internal autonomy and are inclined to be imaginative, unconventional, and disregardful of practical matters.[33] This factor has been repeatedly found to be related to physical fitness.[4, 8, 9, 10, 11]

When comparing the high and low fitness groups that were matched for age, both groups underwent beneficial physiological changes from pre- to post- testing (Table 5).

The multivariate discriminant function analysis demonstrated that the three best personality discriminators (Factors M, H, and L) when combined with the four biochemicals (cholesterol, glucose, testosterone and catecholamines) were capable of significantly ($p < .05$) discriminating between the fitness groups both initially and finally. Both pre- and post- test data revealed that the highly fit individuals were more imaginative, adventurous, and trustful and had lower catecholamine, glucose and cholesterol levels and a higher testosterone level than low-fit subjects.

Factor M has been repeatedly found to be related to physical fitness[4, 6, 7, 8, 9, 10] and in this study it was the most powerful discriminator between the groups in both the pre- and post- test data. Individuals who score high on this factor possess internal autonomy and are inclined to be imaginative, unconventional, and highly flexible in practical matters.[33] Such characteristics appear to be conducive to jogging; likewise, jogging affords a suitable medium for their expression. Many of the high-fit subjects in this study were habitual exercisers who had been jogging for several years. Highly fit individuals also scored low on Factor L indicating an easygoing, friendly relaxed disposition. Similar findings have been reported by Hammer and Wilmore.[5] This factor is related to security and emotional stability[6] and such characteristics have been consistently found to relate to a high physical fitness condition.[7, 8, 10, 13] In this study, high- and low- fit individuals scored within the normal range on Factor H, however, high physical fitness was associated with higher score on that factor indicating that the high-fit subjects tended to be more adventurous, thick-skinned and socially bold. According to Cattell,[33] individuals who score low on this factor have an over-responsive sympathetic nervous system which makes them especially threat-reactive. Neither group was extreme on this factor which may reflect the non-significant difference between the groups on urinary catecholamine excretion rate (Table 6).

At the pre-test, significant differences were found between the two groups on serum glucose and testosterone levels. Lower resting glucose values are regularly found in highly trained individuals[13] and, in this study, reductions were observed in both groups as a result of the exercise program. Neuroticism appears to be related to blood glucose levels[13, 19] and a clear role for testosterone has been established in aggression.[24, 25, 26] Ismail and Young[13] identified a canonical correlation which indicated that individuals with high serum testosterone and glucose concentrations tended to be neurotic and aggressive.

Only recently has testosterone secretion been related to exercise.[11, 44, 45] Young, et al.[11] found that a group of highly-fit young men had a significantly higher level than a similar group of low-fit subjects, but this difference was not significant after an exercise program. They concluded that testosterone level is not purely a function of age but is also dependent upon physical condition, particularly since testosterone is related to other hormones involved in energy metabolism during exercise.[46] Since habitually active individuals differ significantly from low-fit individuals on several personality characteristics,[6, 7, 8, 10, 13] they speculated that these characteristics may have accounted for a suppression of testosterone secretion which was subsequently lifted when the physical fitness of the low-fit subjects increased.

SUMMARY

It was interesting to note that there were no significant biochemical and personality differences between the young and old groups who were matched on physical fitness at the pre-test. Only during the post-tests were significant differences observed (on both parameters) between the two groups. However, when the two fitness groups (who were matched for age) were examined on both biochemical and personality parameters, significant differences were observed at both the pre- and post- tests in favor of the high fit group. Thus, it could be concluded that the biochemical and personality variables were better discriminators between the fitness groups than between the age levels. ☐

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TABLE 1. Normative data of selected physiological variables at the pre- and post- program tests for young versus old participants.

	Pre-test		Post-test		
Variable	$\bar{X}$	S.E.	$\bar{X}$	S.E.	t
Young (n=14) (21-37 yrs.)					
Age (yrs)	31.8	1.2	31.8	1.2	—
Height (cms)	181.1	2.0	181.1	2.0	—
Weight (Kgs)	83.8	5.2	82.9	5.0	1.90
% Lean (body wt)	82.0	1.5	82.3	1.5	1.23
Systolic B.P. (mmHg)	127.1	3.8	124.9	4.0	1.11
Diastolic B.P. (mmHg)	81.4	3.6	79.6	2.4	.87
Maximal H.R. (beats/min.)	176.0	3.3	165.7	3.4	3.41**
VO$_2$ max (ml/kg bd wt/min)	44.6	3.1	42.4	2.4	.72
Physical Fitness Score	341.6	19.8	363.5	14.2	1.42
Old (n=14) (48-61 yrs.)					
Age (yrs.)	51.9	1.2	51.9	1.2	—
Height (cms)	178.7	2.9	178.7	2.9	—
Weight (kgs)	77.6	6.5	77.6	6.5	.07
% Lean (body wt)	82.5	1.8	83.8	1.6	2.72*
Systolic B.P. (mmHg)	130.6	4.3	128.0	3.3	.91
Diastolic B.P. (mmHg)	87.5	3.3	87.4	3.4	.03
Maximal H.R. (beats/min)	168.0	2.5	160.8	3.2	3.06**
VO$_2$ max (ml/kg bd wt/min)	41.0	2.1	44.3	2.6	2.39*
Physical Fitness Scores	330.9	17.2	363.7	16.6	4.34***

*t-Ratio 2.15 ($p < .05$) **t-Ratio 2.98 ($p - .01$) ***t-Ratio 4.22 ($p < .001$)

TABLE 2. Normative Data of the personality and biochemical variables at the pre- and post- program tests for young versus old participants.

Variable	Pre-test $\overline{X}$	Pre-test S.E.	Post-Test $\overline{X}$	Post-Test S.E.	t
Young					
Personality					
Factor E	6.7	.6	6.6	.3	.28
Factor G	7.0	.6	6.4	.6	1.46
Factor M	6.6	.7	5.9	.4	1.38
Biochemical					
Cholesterol (mg%)	196.4	9.4	191.8	13.5	.56
Glucose (mg%)	100.5	6.5	88.4	5.1	2.67*
Testosterone (ng/100ml)	685.2	76.2	688.6	72.1	.04
Catecholamine (ug/24 hrs.)	38.5	3.8	32.3	3.9	1.48
Old					
Personality					
Factor E	5.5	.4	4.7	.4[b]	2.24*
Factor G	5.9	.5	5.4	.4	1.38
Factor M	7.3	.5	7.1	.4[a]	.64
Biochemical					
Cholesterol (mg%)	222.0	10.0	212.0	11.1	1.34
Glucose (mg%)	101.6	3.5	96.7	3.6	1.00
Testosterone (ng/100ml)	617.2	48.5	626.9	43.1	.40
Catecholamine (ug/24 hrs.)	44.1	3.0	46.5	2.3[b]	.72

* t-Ratio 2.15 ($p < .05$)

[a] t-Ratio 2.06 ($p < .05$) (young vs. old)

[b] t-Ratio 2.78 ($p < .01$) (pre vs. post)

TABLE 3. Pre-program discriminant function analysis.

Variable	Coefficients† for Young vs. Old
1. Factor E	1.582
2. Factor G	1.143
3. Factor M	−.612
4. Cholesterol	−.071
5. Catecholamines	−.003
6. Glucose	.050
7. Testosterone	.001
Number of Incorrect Classifications for Each Group	3
Mahalanobis D^2	1.5039
Corresponding F Ratio (d.f. 7,20)	1.16

† All coefficients have been multiplied by 100.

TABLE 4. Post-program discriminant function analysis.

Variable	Coefficients† for Young vs. Old
1. Factor E	3.173
2. Factor G	1.010
3. Factor M	−.537
4. Catecholamines	−.467
5. Glucose	−.290
6. Cholesterol	.004
7. Testosterone	−.002
Number of Incorrect Classifications for Each Group	2
Mahalanobis D^2	4.2655
Corresponding F Ratio (d.f. 7,20)	3.28*

† All coefficients have been multiplied by 100.

* Significant at the .05 level.

TABLE 5. Normative data of selected physiological variables at the pre- and post- program tests for high-fit versus low-fit participants.

	Pre-Test		Post-test		
Variable	$\bar{X}$	S.E.	$\bar{X}$	S.E.	t
High-fit (n=14) (21-61 yrs.)					
Age (yrs.)	41.6	3.0	41.6	3.0	—
Height (cms)	179.4	2.1	179.4	2.1	—
Weight (kgs)	72.5	2.9	72.2	2.7	.71
% Lean (body wt)	86.5	1.2	87.2	1.1	2.97*
Systolic B.P. (mmHg)	120.7	2.0	121.9	2.3	.53
Diastolic B.P. (mmHg)	77.1	1.8	79.7	2.4	1.21
Maximal H.R. (beats/min)	174.9	3.0	167.6	3.0	3.71**
Maximal VO_2 (ml/kg bd wt/min)	50.0	2.0	48.0	2.4	.64
Physical Fitness Score	395.8	9.6	403.2	8.1	.71
Low-fit n=14) (26-61 yrs.)					
Age (yrs.)	42.1	3.1	42.1	3.1	—
Height (cms)	180.5	1.5	180.5	1.5	—
Weight (kgs)	88.9	4.3[b]	88.3	4.1[b]	1.43
% Lean (body wt)	78.1	1.0[c]	78.9	1.1[c]	1.64
Systolic B.P. (mmHg)	137.0	4.3[b]	130.9	4.4	2.58*
Diastolic B.P. (mmHg)	91.7	3.7[b]	87.3	3.4	1.87
Maximal H.R. (beats/min)	169.1	3.0	158.7	3.2	3.13**
Maximal VO_2 (ml/kg bd wt/min)	35.6	1.3[c]	38.6	1.9[b]	2.13
Physical Fitness Score	276.6	7.2[c]	324.0	13.1[c]	4.16**

* t-Ratio 2.15 ($p < .05$)
** t-Ratio 2.98 ($p < .01$)
[a] t-Ratio 2.06 ($p < .05$)
[b] t-Ratio 2.78 ($p < .01$)
[c] t-Ratio 3.71 ($p < .001$)

TABLE 6. Normative data of the biochemical and personality variables at the pre- and post- program tests for high-fit versus low-fit participants.

	Pre-test		Post-test		
Variable	$\bar{X}$	S.E.	$\bar{X}$	S.E.	t
High-fit					
Personality					
Factor H	6.9	.4	6.5	.4	1.17
Factor L	3.1	.5	2.7	.4	1.00
Factor M	8.0	.4	7.2	.4	1.99
Biochemical					
Cholesterol (mg%)	197.6	9.9	185.6	11.8	1.59
Glucose (mg%)	93.7	3.3	88.9	4.4	.99
Testosterone (ng/100ml)	754.3	59.8	707.8	65.5	.93
Catecholamines (ug/24 hrs.)	38.3	3.4	38.5	3.7	.07
Low-fit					
Personality					
Factor H	5.6	.6	5.4	.5	.49
Factor L	4.5	.6	4.6	.5[b]	.41
Factor M	5.9	.6[b]	5.7	.4[a]	.31
Biochemical					
Cholesterol (mg%)	220.8	9.7	218.2	11.9	.33
Glucose (mg%)	108.5	5.9[a]	96.1	4.6	2.63*
Testosterone (ng/100ml)	548.1	55.9[a]	607.6	50.3	.97
Catecholamines (ug/24hrs)	44.3	3.5	40.3	3.7	.88

[a] t-Ratio 2.06 ($p < .05$)
[b] t-Ratio 2.78 ($p < .01$)
* t-Ratio 2.16 ($p < .05$)

TABLE 7. Pre-program discriminant function analysis.

Variable	Coefficients† for High-Fit vs. Low-Fit
1. Factor M	2.769
2. Factor H	1.615
3. Factor L	−.357
4. Catecholamines	−.175
5. Glucose	−.164
6. Cholesterol	−.048
7. Testosterone	.022
Number of Incorrect Classifications for Each Group	3
Mahalanobis D^2	4.5882
Corresponding F Ratio (d.f. 7,20)	3.53*

† All coefficients have been multiplied by 100.
* Significant at the .05 level.

TABLE 8. Post-Program discriminant function analysis.

Variable	Coefficients† for High-Fit vs. Low-Fit
1. Factor M	3.234
2. Factor L	−2.673
3. Factor H	1.966
4. Glucose	−.117
5. Cholesterol	−.026
6. Testosterone	.011
7. Catecholamines	.011
Number of Incorrect Classifications for Each Group	3
Mahalanobis D^2	3.9219
Corresponding F Ratio (d.f. 7,20)	3.02*

† All coefficients have been multiplied by 100.
* Significant at the .05 level.

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USE OF EXERCISE AS A RELAXATION TECHNIQUE*

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It has been reported that ten million Americans suffer from anxiety neurosis and ten to thirty percent of those patients seen by general practitioners are anxiety neurotics.[1] It has also been estimated that 30-70 percent of all patients examined by general practitioners and internists are suffering from conditions that have their origins in unrelieved stress.[2] The potential role of physical activity in the management of anxiety will be reviewed in the present article.

Walking is one of the most frequently prescribed exercises,[3] and for this reason Morgan *et al.*[4] tested the effects of walking one mile at 3.5 mph on the state anxiety of young adult males and females. The subjects were randomly assigned to exercise groups which walked on level grade or 5% grade, and their responses were compared to those of a control group consisting of subjects who simply rested quietly in the supine position. The control, 0% grade, and 5% grade walks resulted in heart rates of 73, 126, and 144 bpm respectively for the women, and 69, 111, and 125 bpm for the men. This particular experiment did not demonstrate an exercise effect on anxiety. One possible explanation would be that exercise might need to be more vigorous in order to demonstrate an effect. A second possibility would be that the anxiety scale employed in this investigation, the IPAT 8-Parallel-Anxiety Battery, was not sensitive to changes since it appears to be more of a trait than a state measure. On the other hand, shifts in anxiety have consistently been demonstrated with this instrument under a variety of circumstances.

Sime's[5] recent research indicating that light exercise does not modify state anxiety corroborates the earlier work[4] reported above. It is also noteworthy that Sime employed the State-Trait Anxiety Inventory (STAI)[6] which is regarded as a more appropriate measure by today's standards. These reports seem to suggest that exercise of a light nature does not alter state anxiety.

The aerobic, or non-lactate producing work of Morgan *et al.*,[4] has been extended to include vigorous exercise.[7] The exercise stimulus in these later investigations produced post-exercise lactate values ranging from 30mg% to 120mg%. The state anxiety scale (STAI)[6] was administered before and following exercise in order to quantify shifts in anxiety. In the first investigation, 40 adult males completed the STAI before, immediately following, and 20 to 30 minutes following a vigorous physical workout lasting approximately 45 minutes. Anxiety was found to increase slightly in the immediate post-exercise setting, but a substantial and significant ($P < .001$) decrease below the base-line level was observed at 20-30 minutes post-exercise. It is also noteworthy that high-anxious individuals experienced a significant reduction ($P < .05$) in state anxiety.

The above findings were replicated in a second study involving 15 adult males who completed the STAI[6] (1) before a vigorous run of fifteen minutes duration, (2) five minutes following the exercise, and (3) 20-30 minutes following exercise. Significant decreases in state anxiety occurred in both post-exercise settings.[7]

Additional evidence supporting the view that exercise may be useful in the management of anxiety comes from the work of Orwin.[8] Orwin has employed exercise to treat agoraphobia, as well as specific phobias. This work seems to suggest that anxiety responses can be inhibited as a result of exercise induced arousal "competing" with the anxiety response. In other words, the perceived autonomic arousal is attributed to the exercise rather than phobic stimuli. It has also been reported by Driscoll[9] that systematic desensitization and exercise plus "pleasant fantasies" are comparable in their ability to reduce anxiety. While exercise or fantasy alone also produced decrements in anxiety, the decreases were not as great as those produced with exercise and fantasy combined. This represents an important therapeutic point when taken in concert with the observation that many distance runners routinely fantasize during training runs as well as competition.[10, 11]

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An exception to the view that exercise reduces anxiety is the position of Pitts and McClure[12] who have proposed that lactate, an exercise metabolite, can provoke anxiety symptoms and attacks in anxiety neurotics. It was reported in a subsequent paper by Pitts "that anxiety symptoms could occur in normal individuals under stress as the result of excess lactate accumulation." It has also been noted by Pitts that the symptoms of anxiety are similar to those produced by physical activity, and exercise may intensify such reactions.[1, 2] The validity of the Pitts-McClure hypothesis of exercise induced anxiety has been evaluated in a series of experiments, and these investigations will now be reviewed.

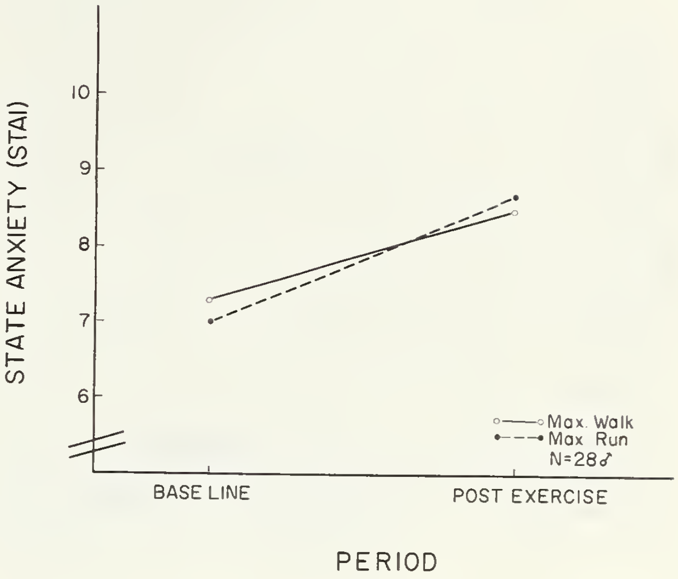

FIGURE 1. State anxiety before and immediately following maximal exercise to exhaustion in the walking and running modes.

EXPERIMENT 1.

The maximal aerobic power (MAP) of twenty-eight adult males was evaluated on a motor driven treadmill on two separate days. The Ss walked at 3.5 mph on level grade, and the treadmill grade was increased 2.5% every two minutes until the Ss reached voluntary exhaustion on the first trial. On the second day, they were required to run at a pace of 6 mph on level grade, and the grade was then increased 2.5% every two minutes until they again reached voluntary exhaustion. The mean heart rate following each trial was 196 bpm, and lactate values ranged from 90 to 100 mg%. State anxiety was evaluated before and immediately following maximal exercise using Spielberger's modified 4-item anxiety scale.[6] The results are summarized in Figure 1. State anxiety was found to increase significantly ($P < .05$) for both the running and walking modes, but the two conditions did not differ from each other. These results tend to support the Pitts-McClure hypothesis of exercise-induced anxiety, but it was felt that immediate post-exercise effects might not be representative of the complete recovery period. Therefore, a second experiment was designed with an aim toward understanding the time course of the anxiety response described above.

EXPERIMENT 2.

The same Ss who participated in the above experiment were tested on two additional occasions. On the first day they walked on the motor driven treadmill at a speed of 3.5 mph, and the grade of the treadmill was such that a metabolic requirement equal to 100% of their previously calculated maximal aerobic power (MAP) was employed. They were evaluated the following day in a similar fashion except that exercise consisted of running at 6 mph on a grade requiring a metabolic cost equivalent to 100% of their previously determined MAP. State anxiety was again evaluated with the 4-item self-report[6] before and immediately following exercise. A third rating of state anxiety was performed within 3-5 minutes post-exercise, and the results are presented in Figure 2. It will be noted that state anxiety increased immediately following exercise in both the running and walking modes just as it had in the first experiment. It is noteworthy, however, that a decrease in this elevated state had occurred within five minutes. In other words, the decrease in state anxiety reported in the earlier experiments took place at 15-30 minutes post-exercise, and this tension reduction response apparently begins within minutes following exercise.

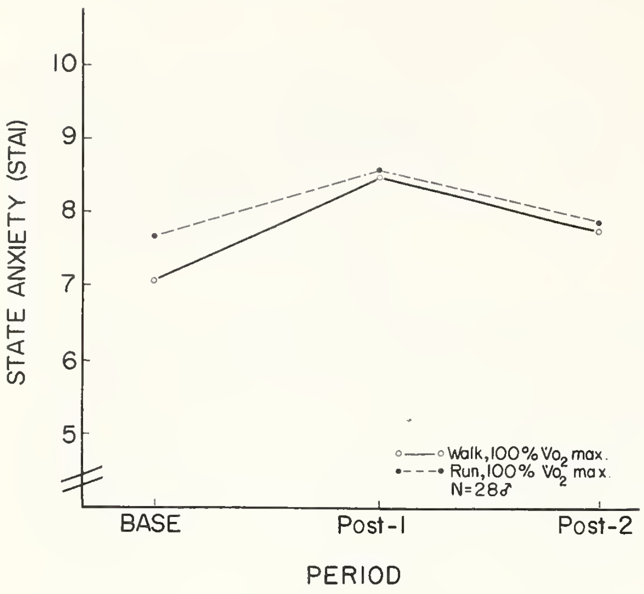

FIGURE 2. State anxiety before, immediately following, and five minutes following maximal exercise performed in the walking and running modes.

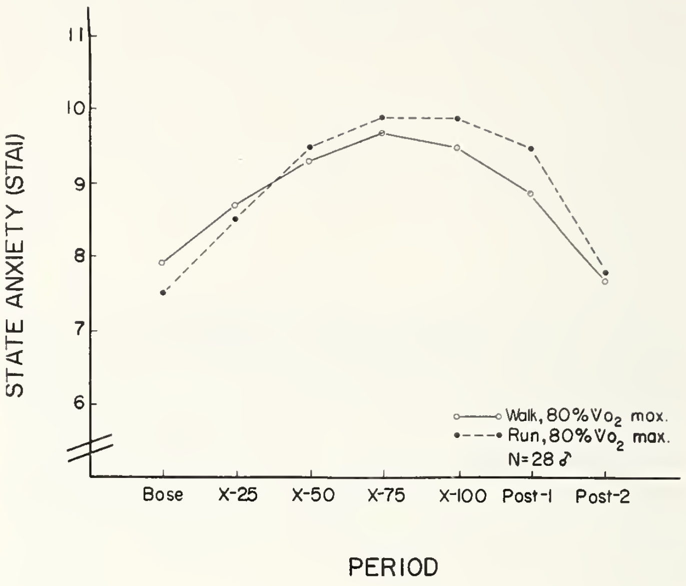

FIGURE 3. State anxiety before, during and 5-10 minutes following exercise in the walking and running modes.

EXPERIMENT 3.

In order to further understand the interaction of anxiety and exercise an experiment was next conducted in which the state anxiety response was evaluated *during* exercise. Verbal self-reports were obtained from the *S*s as they exercised at 80% of VO_2 max. The same group of 28*S*s was tested on two separate days in the walking and running modes, and they exercised to a point of self-imposed exhaustion. The duration of exercise averaged 23 minutes and 24 minutes for the running and walking bouts respectively. Blood lactate increased from 10mg% to 90mg% for both trials, and plasma epinephrine increased about fourfold whereas norepinephrine was elevated sevenfold. Details concerning the catecholamine and lactate assays are described in a recent paper by Horstman *et al.*[13] The four-item state anxiety scale[6] described earlier was administered throughout exercise, and these verbal self-reports are summarized in Figure. 3. It will be noted that state anxiety increased in a fairly linear fashion throughout the first half of the walk and run, reached an asymptote and remained stable for the second half of the exercise, and decreased to base-line levels within 5 minutes following exercise. In other words, the exercise *stressor* (e.g. 80% VO_2 max) provoked a significant elevation in cardiovascular and endocrine responsivity which was accompanied by a significant elevation in state anxiety. This psychobiochemical response was interpreted to represent *eustress* rather than *stress*, however, since an immediate reduction in self-reported anxiety was noted. Furthermore, while we did not evaluate state anxiety 20 to 30 minutes post-exercise, earlier research has consistently demonstrated that anxiety is reduced substantially at such time.[7] Results of an earlier experiment are presented in Figure 4, and it will be noted that a substantial and significant ($P < .01$) decrement in state anxiety occurs at 20-30 minutes post exercise. It seems reasonable to propose that such reductions in state anxiety represent, at least in part, the "exercise high" reported by many habitual exercisers.[14]

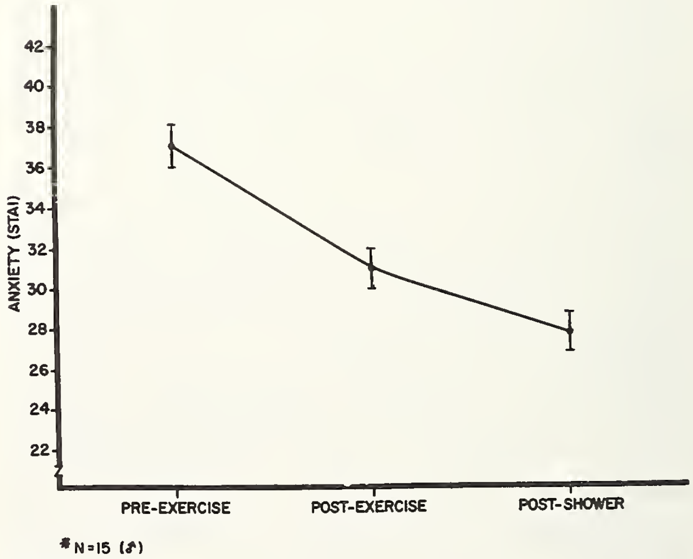

FIGURE 4. State anxiety before and 5-30 minutes following 15 minutes of jogging at a self-established pace.

EXPERIMENT 4.

In order to evaluate the validity of the observations reported above in Experiment 3, a fourth experiment was performed. Twenty-eight (N=28) additional adult males who had not participated in any of the former experiments walked at 80% of maximal aerobic power to self-imposed exhaustion. This group was not quite as physically fit as the former group, and their mean performance time was 18 minutes in contrast to the mean performance of 24 minutes observed for the first group. The psychobiochemical response of this second group, however, was nearly identical with that of the former *S*s. In other words, state anxiety increased significantly with exercise, but there was an immediate reduction in anxiety following exercise.

EXPERIMENT 5.

Many investigators involved in stress research believe that an individual's *perception* of a stressor governs to a large extent the subsequent labeling, definition, or interpretation of the *stressor* as *stress* versus *eustress*. This particular view rejects simplistic "stimulus-response" (SR) models of stress and argues for the more scientifically defensible notion of "stimulus organism-response" (SO-R) models. The efficacy of the SO-R model has been repeatedly supported by a number of speakers at the present symposium, and such a model argues that responsivity is mediated in part by cognition. It is almost as though psychology, after many years, has regained its *mind*!

In order to evaluate the extent to which perception influences the anxiety response to exercise, a fifth experiment was performed. In this investigation, 18 adult males were randomly assigned to either (1) a standard or control condition (N=6); (2) a placebo group (N=6); or (3) a meditation group. The *S*s in the control condition exercised at 80% of maximal aerobic power to self-imposed exhaustion just as *S*s in the previous experiments had done. A second group of *S*s (N=6) received a lactose capsule with instructions to the effect that they would not experience as much fatigue and discomfort as they had when previously performing the same task. The third group (N=6) of *S*s were instructed in the relaxation response developed by Benson,[15] and they were informed that practice of this form of meditation would result in reduced fatigue and discomfort as they exercised. The results of this experiment are summarized in Figure 5.

Inspection of Figure 5 reveals that the control group experienced a significant increase in state anxiety as measured by self-report;[6] this was followed by an immediate reduction in anxiety following exercise. This observation was replicated in each experiment cited above. It will be noted, however, that the typical exercise-induced anxiety response was arrested in both the placebo and meditation groups. Therefore, the view that response to a given stressor is mediated in part by one's perception or cognition is supported. It is also noteworthy that the three groups did not differ in their cardiovascular or endocrine response to the stressor.

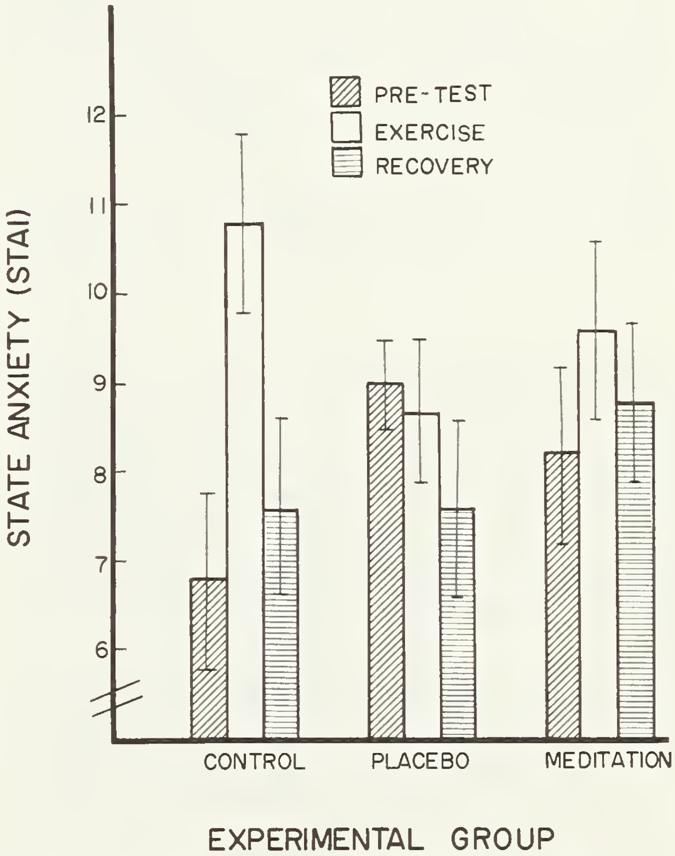

FIGURE 5. State anxiety before, immediately following, and five minutes following an endurance run at 80% of VO_2 max under control (N=6), placebo (N=6), and meditation (N=6) conditions.

DISCUSSION

The experiments summarized in this paper can be viewed as refuting the Pitts-McClure hypothesis of exercise-induced anxiety. While state anxiety has been observed to increase with high intensity exercise, it has been consistently demonstrated that anxiety decreases rapidly following such exercise. Furthermore, tension levels actually fall substantially below pre-exercise values when anxiety is evaluated at 15-30 minutes post-exercise. The reduction in anxiety has been observed to follow vigorous exercise in normal as well as clinically anxious individuals.

It is possible that the major psychologic benefits of vigorous physical activity reside in its ability to maintain a desired tension state. It is quite conceivable that daily exercise results in tension reduction which is followed by a resurgence of anxiety across the subsequent 24-hour period. If, in fact, anxiety and physical activity were to covary in such a manner, the primary psychologic benefit of exercise would be its preventive power in terms of positive mental health. An important theoretical and applied issue which has not been addressed however, relates to the important question of whether or not exercise *causes* tension reduction. The tacit assumption seems to have been that decreases in anxiety were preceded by exercise, and therefore, exercise *caused* the tension reduction. In point of fact, however, there has been little if any evidence presented to support the notion of causality. If one were to assume that anxiety was reduced by exercise, another important question would be the extent to which such a treatment was more efficacious than other treatments. It would be of major theoretical and applied importance if exercise were no more effective in reducing anxiety than "passive" therapies such as progressive relaxation, meditation, hypnosis, biofeedback, and so on.

The importance of the *causality* vs. *association* issue has been reinforced in several recent papers involving anxiety management. It has been demonstrated by Michaels *et al.*,[16] for example, that transcendental meditation (TM) is *associated* with a decrease in plasma epinephrine, a biochemical measure of anxiety. Hence, it would be easy to jump to the conclusion that TM *causes* anxiety or plasma epinephrine to decrease. However, the control group in their investigation[13] experienced comparable reductions in plasma epinephrine and norepinephrine. This, of course, raises the classic question of causality, and it forces one to conclude that while TM may reduce plasma catecholamines, *it is no more effective in doing so than a simple rest in a quiet room!*

In a somewhat related investigation, deVries *et al.*[17] compared the effectiveness of relaxation training and biofeedback in reducing tension as measured electromyographically. A rest break was also employed in this study as a control, and it was found that EMG activity was reduced just as much with this simple rest period.

Recent psychometric evidence has been presented which corroborates the above biochemical and neurophysiological data. In this more recent investigation, Bahrke and Morgan[18] randomly assigned 75 adult males to conditions consisting of twenty minutes of (1) exercise at 70% of maximal aerobic power, (2) non-cultic meditation, or (3) a control condition involving quiet rest in a sound-filtered room. The exercise and meditation groups both experienced significant decreases in state anxiety as expected. An unexpected result, however, was the observation that the control group also experienced a decrease in state anxiety. A control or placebo group was employed in an attempt to quantify the Hawthorne effect, or *any treatment* versus *no treatment*. These biochemical,[16] neurophysiological,[17] and psychometric[18] findings have led Bahrke and Morgan[18] to speculate that *diversion*, not exercise, biofeedback, or meditation is the crucial ingredient. In other words, it appears that "time out," or simple rest breaks, may be effective in the management of anxiety. A common clinical observation is that many distance runners spontaneously report that running permits them to "drift away from the concerns of the day" and become deeply relaxed as a consequence.[10] Needless to say, exercise involves many physiological changes which are not observed with biofeedback, relaxation training, meditation, or "time out" therapy. For example, substantial increases in core temperature, cardiac frequency, systolic blood pressure, ventilatory minute volume, and catecholamine production accompany vigorous physical activity whereas the converse occurs with aforementioned "passive" therapies. Furthermore, it is possible that vigorous exercise may also alter brain biochemistry in such a way as to *cause* improved affect. The important point is that *causal* evidence has not been presented. This represents a significant methodological issue in view of the observation that quiet rest breaks or "time out" are associated with decrements in anxiety as measured biochemically, neurophysiologically, or by self-report, which equals *quantitatively* reductions in anxiety following meditation, biofeedback, or exercise. Whether or not reduced anxiety following "time out" therapy is *qualitatively* equal to more traditional approaches such as exercise has not yet been addressed. Also, generalizability of this experimental research to the clinical setting has not been examined.

SUMMARY

The present clinical and experimental evidence overwhelmingly supports the view that vigorous physical activity of an acute nature is consistently *associated* with a decrease in state anxiety. This generalization holds for both normal, as well as clinically anxious individuals. The view that lactate, an exercise metabolite, produces anxiety has been convincingly refuted at both a theoretical and experimental level. While the tacit assumption seems to be that exercise *per se* causes anxiety to be reduced, there has simply not been evidence presented to support *causality*. ☐

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NOTES

1. The views, opinions, and or findings contained in this report are those of the author(s) and should not be construed as an official Department of the Army position, policy, or decision, unless so designated by other official documentation.
   Human subjects participated in these studies after giving their free and informed voluntary consent. Investigators adhered to AR 70-25 and USAMRDC Regulation 70-25 on Use of Volunteers in Research.
2. The authors are indebted to Damon Burton, Domenic Castignetti, Dan Ebert, Vincent Forte, and Adrian Lussier for their excellent technical assistance.
3. Present address: Department of Physical Education and Dance, Sport Psychology Laboratory, University of Wisconsin, Madison, Wisconsin 53706.

SUMMATION: THE SECOND NATIONAL CONFERENCE ON EMOTIONAL STRESS AND HEART DISEASE

ROBERT S. ELIOT, M.D.*

Emotional stress is a very popular concern in today's professional and nonprofessional world. Recently, its importance has been promoted to the public. Many of the awareness activities have been in the hands of kooks, cults, and polycults. Their efforts have made it extremely difficult for those who wish to provide credible information for review by proper authorities. There is little question but that the kooks have done ethical professional investigators a considerable disservice in the development of this field. The question we should ask, perhaps, is who should really be representing education, service, and research in the interest of medicine, its allied health professions, and the public. Fortunately, it is meetings like this that help to develop a population of credible voices concerned not so much with the demagogic "who is right" or with the dogmatic "I say, you do," but instead with the pragmatic "what is right."

Unquestionably today, investigators in the field of emotional stress and cardiovascular disease find themselves together walking a metaphorical tight rope. They are moving from a state of relative ignorance toward new knowledge. Let us pay close attention to our goals so that we do not lose our balance as we move from one end of this tight rope to the other. There is little question that each step must be carefully taken.

This tight rope was assembled and put into place by a small group of individuals who were concerned with the need to evaluate the role of emotional stress in cardiovascular disease. The South Carolina Heart Association and others who joined them in this effort were clearly responsible for continuing to stimulate the interest of our profession and its major organizations in the development of credible work and useful information in this critical area.

One of the great challenges ahead which will require scientific and technologic systems is the matter of quantitating biobehavioral risks. Certainly there is no more difficult challenge than that of putting numbers on sociobiologic events. Once indices become apparent, I am confident they will be utilized by those who are conservative if not dubious with regard to the potential practical value of this field.

We are aware that there are powerful economic factors operating with regard to the entire field of medicine and that these will influence the interest and enthusiasm for development of biobehavioral medicine and health promotion programs. For example, the return to work following myocardial infarction is more frequent than that following coronary artery bypass surgery. The reasons for this observation need to be identified. It is likely that the answer will be complex and, it will most likely involve psychosocial dimensions and issues.

Assessment and intervention in either of the aforementioned cases is not simple. It must be comprehensive and take into consideration all aspects of the patient's life. Who is this person? Where is he coming from and where is he going as a total psychobiological unit? Can there be such a thing in the future as a Center for Behavioral Modification which addresses itself to these complex forms of management? Undoubtedly the blending of science and technology with the behavioral aspects of medicine may create such opportunities and, indeed, bring into existence centers that focus on the entire picture of the patient's illness, in balance with his lifestyle.

Repeatedly, throughout this meeting we have heard various investigators point to the necessity of determining individual perception. It is the individual's self opinion and appraisal, and not that of his peers, which appears critical in the development of coping mechanisms which are either constructive or self-destructive.

To help in this field, we need young investigators. I was very pleased to see that a good percentage of the people who presented papers at this program were young, well motivated, and stimulated. Their input will be necessary to assure a continuing flow of creative input to a rapidly developing field. It is also my sincere hope that as we develop we will not become an impenetrable aristocracy, absolutely insensitive to the need for change, for further development, for constant re-evaluation, and for the acceptance of new ideas from those not yet prejudiced by the stereotyped formats of their original medical training.

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* Professor and Chairman, Division of Cardiology, University of Nebraska Medical Center, Omaha, Nebraska 68105.

In a nutshell then, we have agreed to begin some practical implementation. We have seen some parts of this field that may serve as adjuncts to other specialties and that are deserving of further careful animal or human investigation. When evaluation has been completed, some of these features will find themselves in practical clinical medicine settings. It will be years before we know whether some of them will prolong lives, but it seems to me that many of these developments could enhance the quality of life. As long as the methods are safe and sensible, we should find them welcome adjuncts to caring when caring is a less available option. Keeping people healthy and productive without being self-destructive is the battle cry for today's national and economic world war. It is conferences such as this that will help to forge weapons for tomorrow's battle for health promotion. It is one war we can wage with malice toward none.

Over the past three days, we have learned that animal studies have provided some very important directions for future clinical research. These animal studies have offered observations that would take a lifetime in human subjects. We don't seem to have that much time. Granted, animal experiments are not human ones, yet we can profit by viewing animal studies more for their similarities than for their differences. Indeed, it is these similarities between animals and humans that allow us a perspective with regard to what directions we might pursue in a clinical sense. It seems that the myocardium of the mouse can be the victim of social consequences and many of us have the suspicion that this is true in man as well. This seems especially likely in the realm of sudden death, myocardial infarction, and potentially fatal arrhythmias. Some of the myocardial histologic features of sudden death found in coroners' cases can be duplicated in animal experiments. It is of further interest that these changes are either prevented or remarkably diminished by beta blockade, again implying that the autonomic nervous system and sympathetic overdrive play a powerful role in the field of myocardial necrosis, both in animals and man.

We have reviewed the psychosocial influences and pathways that can lead to hypertension and we have discussed the possibility of behavioral or personality variables in these people. We are impressed that much remains to be done in this area but there are some important observations that have been made regarding such important areas as the pathophysiology of hypertension for example. Stress is clearly a common thread in the complex fabric of hypertension.

Biobehavioral influences have now been identified both in hypertension and in coronary heart disease. We now know, for example, that Type A behavior can enhance the multivariate risks in coronary heart disease. Such coronary prone behavior is a statistically significant and measurable phenomenon; it probably has a great number of components, subcomponents and overlapping dimensions. These are in need of further clarification. At this point in the development of the field, however, it is not surprising that further work is necessary. It will offer a continuing challenge to researchers. We also need to consider how this behavioral pattern interacts with the lifestyle and environment of the individual. Is this interaction optimal or self-destructive? Obviously, there are many Type A's who escape into senescence without coronary heart disease. The reasons for surviving as well as succumbing require further elaboration, individualization, and clarification. It thus remains difficult for us to select the Type A individual who is headed for disaster.

We would like to find methods that more selectively identify the individual who carries the highest risk for the immediate future. These efforts are in process. Both written and oral interview techniques have more clearly pointed the direction toward identifying such persons at risk. One dimension which may offer better selectivity is that form of study which provokes the individual physiologically as well as psychologically. Attempts to characterize, to qualify, and even to quantitate these responses may assist in differentiating between the Type A who is resourceful and resilient as compared to the more self-destructive Type A engaged in the joyless struggle (Sisyphus complex). Preliminary studies aimed at detecting such individuals at high risk have been presented at this meeting. These studies attempt to correlate and standardize physiological and psychological responses with a standardized provocative psychological input. Our appetites have been whetted for more comprehensive evaluations of this type.

The role of intervention has been touched upon over the past three days. The selective use of biofeedback techniques has been received with a sense of cautious practical credibility in the hands of two different groups, each of considerable repute. In one study, data suggests the control of Raynaud's phenomenon; in the other, results suggest some success in the control of hypertension. In the hands of one investigator, there was difficulty in the management of hypertension employing biofeedback techniques; with the other there was evidence of some success. The disparity in success may in part relate to how much self mastery and control the patient feels he has over his own therapeutic management as compared to the amount of control by the physician. Adherence was best achieved when the patient was given the responsibility of managing most of his own therapy. It might well be expected that this approach will prove to offer better patient adherence, whether it be in biofeedback or other therapeutic avenues. The approach deserves careful and continuing investigation and evaluation.

Exercise is a popular, fascinating and highly complex dimension of intervention, rehabilitation, and health promotion. It is so complex that an in-depth meeting could be developed on this subject alone. Nevertheless, it was interesting to note the metabolic and physiologic differences observed between the fit and the unfit. One wonders how much difference there is on a genetic basis. We realize we can breed either race horses or plug horses. Undoubtedly, many humans function like race horses without a great deal of obvious exercise conditioning. Thus, one must consider a potent genetic component in physical fitness, and its interrelationship with metabolic, physiologic, and other factors. Unquestionably one man's meat may be another's poison. As was pointed out in the studies on control of anxiety employing exercise, it was remarkable to note that exercise, meditative techniques, or simply sitting in a quiet room all had salutary effects on the level of anxiety. The control of anxiety by any technique is welcome in contemporary society. We have utilized the concepts of "time out," time out from the game of life that is. It may well be that exercise for some offers "time out," whereas transcendental meditation or just sitting quietly is effective for others. Any technique which will temporarily detach the individual from his arousal and tenacity to lifestyle and occupation should be of assistance with the control of this contemporary preoccupation. In the area of hypertension, however, and in some other cardiovascular conditions, exercise appears extremely effective when individually prescribed and appropriately implemented.

The area of behavioral medicine is deserving of comment as well. It is perhaps better referred to as biobehavioral medicine as this includes all medical and behavioral sciences. It is not viewed as a field in and of itself, but instead, as an adjunct to any health related speciality. As Doctor Weiss has put it, it is a joining together. There is thus something for everyone in the field of biobehavioral medicine and in the utilization of the appropriate techniques of behavioral modification.

It is a clear need that the important scientific and technologic advances of medical science be blended and utilized for the further development of the field of biobehavioral medicine. The current overbalance on the side of technology should serve as a stimulus to those who seek to develop the humanistic aspects of medicine. Thus, the field is not viewed in an exclusive, but instead, in an inclusive fashion. □

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President's Page

THE ANNUAL CONVENTION

One of our priorities for 1979 was to re-study the effectiveness of our annual meeting. This was brought about by an apparent lack of interest on the part of our general membership in attending this annual gathering. When considering the cost involved, the event is non-productive in terms of direct benefits to the membership. What can be done to improve this participation?

The Kentucky Medical Association offers a glaring example of success in terms of percentage of practicing physicians who are members and in terms of attendance at their annual meeting. Some 40 to 45 percent of their members attend the annual meeting, while only some 13 percent of our members attend the SCMA convention.

On September 25-27, 1979, Dr. Euta Colvin, Dr. Halsted Stone, Mr. Blake Williams and I attended the Kentucky Medical Association meeting in Louisville, Kentucky. This convention is held in a convention center in a major city (usually Louisville) at the site of the major physician population. The convention center is of such size as to allow adequate space for exhibitors (commercial and scientific) and so designed as to make it necessary for physicians to pass through the exhibitor area in going to the scientific lectures and displays. There is a waiting list of exhibitors based on excellent participation, and income derived from leasing of space is a major source of income to cover the cost of the convention.

The scientific program is a joint project by specialty societies and KMA. Each year, a theme is developed (i.e., emergencies or trauma) and programs are developed around this theme. Each morning, topics of general interest are presented, Monday through Thursday, while the afternoons are devoted to specialty items within the theme. Specialty speakers for the specialty societies present in the morning, and part of their honorarium is paid by KMA.

The House of Delegates convenes on Sunday afternoon, and the final session is held Wednesday evening, beginning at 6:00 p.m. On Monday night, a KMPAC (the KMA political action committee) dinner is held where two speakers of opposing parties debate an issue or present a program. On Wednesday, there is a luncheon honoring visitors and dignitaries. The installation of the President is held at this time, and, in addition, the Physician of the Year is recognized and receives an award.

The Alumni Association of the two medical schools prepare separate agendas and distribute them to their alumni, and they work diligently to encourage large attendance.

This plan, as held at the KMA, will be presented to the Hilton Head Leadership Conference by staff from Kentucky. Hopefully, a similar plan can be developed for the SCMA Convention. The success of this event will depend on the commitment given by the specialty societies, and the support this Association can give to these societies, plus a good campaign to advise our membership. The many details required to bring about a change of this magnitude may prevent full implementation in 1980. We'll keep working to make the Annual Convention THE place for men and women of medicine to meet to fulfill our resolves and to become a united force for the common good.

Any suggestions?

Harrison L. Peeples, M.D.,
President

"Better Health Through Health Education"

SOUTH CAROLINA MEDICAL ASSOCIATION

AUXILIARY

HEALTH CAREERS FAIR

The Ninth Annual Health Careers Fair will be held February 13 and 14, 1980 from 10:00 a.m. to 4:00 p.m. It will be presented by the South Carolina Medical Association Auxiliary at the Municipal Auditorium in Charleston.

Some of the presentations at the Fair include demonstrations, exhibits, counseling, and scholarship information. Tours of the Medical University and Allied Health Schools are being arranged for interested students. In the past, over 18,000 Junior and Senior High School students from at least 70 schools in 22 counties have visited the Fair.

Mrs. William J. Fogle is serving as Chairman of the Health Careers Fair for this year.

Mrs. William Fogle, Health Careers Fair Chairman, left, and Mrs. Arthur LaBruce, President-Elect and past Health Careers Fair Chairman.

Exhibits at 1979 Health Careers Fair.

AMA Exhibit of transparent twins showing skeletal system and body organs.

Stryker bed frame used particularly for burn patients.

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SOUTH CAROLINA PESTICIDE POISONINGS, 1978*

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The first morbidity statistics reporting pesticide poisonings in South Carolina were gathered by Pietsch, et al[1] (1968). This work, which consolidated poisoning reports from the S. C. State Health Department, from investigations by the U. S. Department of Agriculture, and from the state's two Poison Control Centers in Charleston and Columbia reported that 600 cases of all poisonings occurred annually with 60 of these being pesticide related. This figure, however, was found to be inaccurate when compared to subsequent pesticide poisoning surveys of South Carolina physicians.

Keil, et al[2] (1970) and Whitlock et al[3] (1972) surveyed those physicians in South Carolina who were in a specialty or practice which were more apt to encounter pesticide poisonings. Both surveys queried approximately one thousand practicing physicians with the 1970 study reporting 572 pesticide poisonings occurring in 1969. The 1972 study reported a 10% increase in pesticide poisonings to 627 cases for 1971. A third physician survey[4] was conducted in 1974 by the South Carolina Pesticide Epidemiologic Studies Center. This study estimated that pesticide poisonings decreased by 10 percent to 570 cases in 1973.

The purpose of the study described in this report was to document shifts in pesticide morbidity and distribution from the previous surveys. Additionally, it was believed that this survey would also serve as an indicator of the effectiveness of newly enacted pesticide laws since the last survey such as applicator certification, restricted use and cancellation of particular pesticides and child resistant packaging of pesticide products.

METHODS:

One thousand one hundred and seventy-three physicians whose practice or specialty, as listed in the 1978-79 directory of the State Board of Medical Examiners of South Carolina,[5] included emergency medicine, family or general practice, occupational medicine and pediatrics were selected for the survey. A letter was sent to each of these physicians which explained the objectives of the survey and requested their cooperation through answering two questions on an enclosed stamped postcard. The information requested included the number of pesticide poisoning cases seen or treated in the last 12

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** Research Analyst

*** Professor of Medicine in Family Practice months and their opinion as to whether the incidence of pesticide poisonings was increasing, decreasing or remaining the same.

The medical specialties selected for the survey and the information requested for the 1978 study were identical to the three previous physician surveys. The study population was surveyed during the last quarter of 1978 and the majority of responses were received by late January 1979.

RESULTS AND DISCUSSION:

Four hundred and fifty-three physicians responded to the survey and reported they had seen or treated 505 cases of pesticide poisoning during the previous 12 months. This represents an 11% decrease in the number of cases from the 718 physicians who reported 570 pesticide poisonings during 1973. Forty-two percent of the respondents believed that the frequency of pesticide poisoning was remaining the same while 21 percent thought the number of cases was decreasing. Only nine percent of the physicians indicated an increase in the number of cases and 28 percent of the respondents had no opinion.

The 11% decrease in poisonings reported in this survey was accompanied by a corresponding decrease in responding physicians as compared to the previous study. Follow-up of the non-respondents to increase the response rate of the current survey was not considered. It was thought that this procedure would introduce bias since follow-up was not utilized in the other studies. An indicator of the survey population response is the ratio of pesticide poisonings per responding physician. The 1978 ratio (1.11) was .32 greater than found in 1973. This suggests that the 1978 respondents were more likely to be those physicians who actually observed pesticide poisonings and that those physicians who did not see any cases were non-respondents.

Table 1 lists the reported number of pesticide poisoning cases by county and survey year. Also included in this table is the physician response by county for 1973 and 1978. During 1978, Charleston County physicians reported the highest number of cases (n = 75). The next two leading counties for pesticide poisonings include Orangeburg (42) and Horry (33). These counties respectively led the State in 1978 production of soybeans and tobacco;[6] crops which require diverse pesticide applications for maximum yield.

Table 1

South Carolina Physician Diagnosed Pesticide Poisonings
by County and Survey Year

		Number of Cases Reported		
County	1969	1971	1973	1978
Abbeville	1	13	1 (62)*	1 (57)
Aiken	2	20	5 (41)	12 (46)
Allendale	20	2	0 (33)	0 (33)
Anderson	9	13	10 (69)	15 (46)
Bamberg	5	8	8 (60)	9 (83)
Barnwell	1	13	3 (63)	5 (50)
Beaufort	11	14	16 (55)	17 (27)
Berkeley	3	6	4 (63)	1 (71)
Calhoun	10	1	4 (66)	6 (33)
Charleston	12	22	44 (58)	75 (45)
Cherokee	10	7	23 (55)	1 (25)
Chester	2	8	1 (67)	0 (43)
Chesterfield	5	1	3 (33)	3 (27)
Clarendon	10	18	13 (75)	2 (29)
Colleton	5	13	17 (43)	6 (58)
Darlington	22	13	24 (54)	27 (66)
Dillon	14	11	3 (66)	3 (22)
Dorchester	4	0	1 (44)	5 (33)
Edgefield	8	4	5 (83)	4 (66)
Fairfield	12	9	2 (67)	3 (40)
Florence	54	35	65 (61)	14 (39)
Georgetown	1	3	0 (61)	19 (53)
Greenville	72	32	24 (46)	29 (38)
Greenwood	6	12	2 (50)	4 (19)
Hampton	1	12	2 (57)	0 (14)
Horry	54	54	51 (53)	33 (32)
Jasper	0	1	1 (50)	7 (66)
Kershaw	11	9	6 (33)	1 (16)
Lancaster	2	3	8 (60)	1 (41)
Laurens	4	10	6 (58)	1 (40)
Lee	6	3	3 (66)	7 (75)
Lexington	22	24	46 (25)	6 (38)
Marion	19	56	14 (47)	23 (43)
Marlboro	5	8	19 (53)	11 (44)
McCormick	0	0	1 (50)	0 (25)
Newberry	0	2	1 (33)	4 (13)
Oconee	23	6	12 (68)	4 (37)
Orangeburg	30	49	32 (61)	42 (58)
Pickens	2	10	1 (44)	0 (14)
Richland	26	26	14 (43)	24 (28)
Saluda	2	2	2 (66)	0 (25)
Spartanburg	21	41	38 (43)	25 (41)
Sumter	4	18	13 (56)	16 (52)
Union	1	0	1 (44)	0 (22)
Williamsburg	12	2	8 (55)	23 (67)
York	28	13	13 (65)	16 (32)
	572 (57)	627 (61)	570 (52)	505 (39)

*Percent of physicians responding are shown in parentheses.

Florence County recorded the largest decrease in poisonings with 51 fewer cases than found in 1973, however, only twelve of the thirty-one physicians surveyed in this county responded to the study. Allendale, Chester, Hampton, McCormick, Pickens, Saluda, and Union Counties reported no pesticide poisonings during 1978 and these same counties each reported two or less during the last survey.

Figure 1 presents the distribution of the reported pesticide poisonings by congressional district. District VI, traditionally recognized for its prominent tobacco and cotton farming, reported the largest number of cases (160) and has led all other congressional districts in the last three surveys. District V, accounted for the largest decrease in cases with 29 fewer poisonings than reported in 1973. The greatest increase in cases is found in District I with 15 more poisonings than practitioners reported in the last survey.

Table 2 lists the aggregate county pesticide poisonings of congressional districts per 100,000 population. Little difference has been observed in the overall state rates. A slight downward shift is seen from 1971 through 1978, although this

Table 2

South Carolina Pesticide Poisonings
Per 100,000 Population[1] by
Congressional Districts

Congressional District	1969	1971	1973	1978
I	14	18	21(57)[2]	24 (42)
II	22	28	25(48)	21 (36)
III	12	21	9(55)	10 (35)
IV	21	18	15(45)	12 (39)
V	18	17	18(58)	11 (34)
VI	49	48	49(58)	42 (45)
State	22 (58)	25 (61)	22(52)	20 (39)

[1] Population based on 1970 U.S. Census.

[2] Percent of physician responding are shown in parentheses.

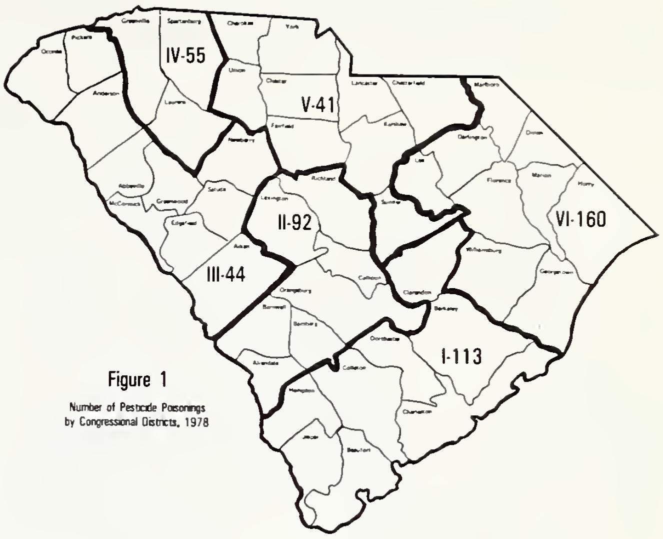

Figure 1

Number of Pesticide Poisonings by Congressional Districts, 1978

may be a contributing influence of the declining physician response. Two distinct trends are apparent through the four survey years. First, the rates of Congressional District I have steadily increased with a 70% change being observed in 1978 as compared to 1969. Conversely, the rates of Congressional District IV have progressively decreased over the years of study with a 43% decline being observed by 1978. Congressional District VI has maintained the highest rates throughout the four surveys and continues to show a rate approximately twice that of the next highest district and the state for any survey year.

SUMMARY:

A survey of 1,173 South Carolina physicians indicated that 505 pesticide poisonings were observed by the 453 respondents during 1978. This represents an eleven percent decrease in cases since the 1973 survey. The rate per 100,000 population of Congressional District VI remains high and is almost twice the rates of the state and the next highest district. Forty-two percent of the respondents believed the frequency of pesticide poisonings was remaining the same while 21 percent thought the number of cases were declining. Only nine percent of the physicians thought there was an increase in the number of cases.

The incidence of pesticide poisonings in South Carolina has changed little over the survey years. The observed rate based upon the physician response to this survey is 20 per 100,000 population as compared to an average of 23 per 100,000 found in the three previous studies. The slight decline in both the number and incidence of pesticide poisonings may be due in part to the lower physician response rate. Other parameters to consider include a greater awareness of pesticide hazards among the public and physicians as well as the enactment of regulations which promote the safe usage and storage of pesticides. ☐

DISCLAIMER STATEMENT

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EXPERIENCES WITH SWAN-GANZ CATHETERS IN A 70 BED COMMUNITY HOSPITAL*

DOUGLAS W. JENKINS, M.D.**

Having read the recent literature as to the value of the Swan-Ganz catheter in managing critically ill patients[1, 2, 3, 4] and having little confidence in the information gained from a central venous pressure (CVP) line, we decided to attempt the institution of Swan-Ganz catheters at our hospital. We report here our results with the first twenty insertions and demonstrate that the Swan-Ganz catheter can be utilized in a community hospital environment.

We initiated the usage of the Swan-Ganz catheter in October 1978 using a standard monitoring kit with a continuous flush valve and disposable transducer domes. We also added 1500 units of heparin to the 500cc bag of fluid and 3,000 units of heparin to the 1000cc bag of fluid with 300mm of mercury pressure applied to the bag with a pressure cuff.

We were able to enter the pulmonary artery in 19 of the 20 patients which gave us a 95% insertion rate. In one patient, we inserted the Swan-Ganz catheter twice after having to remove the original catheter because of inadvertently cutting it after it was in place. The second catheter was inserted in the right internal jugular as was the first.

Fluoroscopy was not used with any of our insertions; however, we did not find it necessary as the pressure curve on the oscilloscope was quite adequate in giving us the location of the catheter tip, i.e., right atrium, right ventricle, and pulmonary artery. We used three sites for introduction of a catheter (right internal jugular, right subclavian, and right antecubital fossa) and found that the internal jugular was the most satisfactory. The internal jugular site produced the most stable catheter and generally resulted in the quickest insertion time. The antecubital fossa was entered via a cut-down, while a Sorenson introducer was used to enter the subclavian and internal jugular veins. We had no complications from entering these sites with the above methods.

The time taken to actually insert the catheter varied from 2½ hours to 15 minutes. The quickest time of insertion was with the Sorenson introducer via the internal jugular, with the longest being via cut-down in the antecubital fossa. We also noted a quicker and easier insertion with the 7 French catheter as opposed to the 5 French catheter.

All of our Swan-Ganz catheters were inserted at the bedside and the position was confirmed by chest x-rays. In our series the pulmonary artery diastolic pressure and pulmonary capillary wedge pressure correlated as has been noted previously.[5]

The one patient in whom we were unable to insert the catheter had a gram negative sepsis and we were able to enter the right ventricle but never could enter the pulmonary artery. This was probably due to his rather poor clinical condition with a poor cardiac flow rate. Two blood cultures were subsequently positive for salmonella and he remained markedly hypotensive throughout his hospitalization.

We had one case of catheter displacement back into the right ventricle which occurred with a catheter inserted in the antecubital fossa, and one case of a catheter coiling in the right ventricle. The catheter that coiled in the right ventricle was easily removed without knotting and probably could have been avoided if more attention was noted to the distance of insertion as marked on the catheter. These two problems occurred with the first five insertions. We also noted that catheter stability was a problem only when the antecubital fossa was used as the site of introduction.

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Of the twenty patients, ten subsequently died of their original disease. Their deaths were not related to the catheter insertions. Four of these patients who died were septic with positive blood cultures noted prior to insertion of the catheter, and an additional two were clinically septic although blood cultures were not positive. An additional two patients were "code blue" patients having received CPR prior to being admitted to the hospital. These two patients were successfully defibrillated and subsequently died from central nervous system complications. The final two deaths were attributed to irreversible congestive heart failure having been proved by persistently elevated pulmonary artery pressures in spite of vigorous diuresis and usage of after load reduction and inotrophic agents.

In the ten patients who survived their illness, the Swan-Ganz catheter was invaluable in reassuring us that their management was correct as regards transfusions, after load reduction, inotrophic agents, or simple fluid administration. To break this down further, two patients were monitored post-operatively, one with abdominal aneurysm resection and the other an elderly male with a mesenteric infarct. In both patients, fluid and blood replacement were of paramount importance. The other eight patients were those of congestive heart failure with complications such as anemia, hypotension, or failure to respond to diuretics. All were managed according to the dictates of the information obtained from pulmonary artery pressure.

In conclusion, we have found the Swan-Ganz catheter to be quite helpful in our community hospital and thus far associated with few complications. The placement of the catheter can readily be done when no fluoroscopy is available as bedside oscilloscope monitoring gives accurate information as to its location. We anticipate that it will be used more widely than has been the CVP line in the past. ☐

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THE LOW-BIRTH WEIGHT INFANT: OBSTETRICAL PERSPECTIVES*

RONALD V. WADE, M.D., F.A.C.O.G.**

The vast majority of the perinatal losses in the United States today are made up of low birthweight infants. It is entirely fitting that special attention be paid to this group of patients by all health care professionals and lay persons interested in lowering the perinatal mortality rate. In order to study this problem in some detail and for discussion purposes, we should really discuss two fairly distinct, but occasionally coexistent entities; the average weight for gestational age baby who happens to be born prematurely and the small for gestational age baby who for some reason has experienced inadequate intrauterine growth.

Premature delivery is the single greatest hazard that an unborn child faces. The underlying pathophysiologic mechanisms that result in premature labor are largely unknown. Specific obstetrical situations associated with premature labor are summarized in Table I. Unfortunately a large proportion of the cases of premature labor have no predictive factors to allow one to anticipate this complication.

Several pharmacological modalities are now being used to arrest premature labor. These drug regimens revolve around the accepted concepts of the physiology of uterine contractions. It is now felt that uterine contractions are mediated through action of the prostaglandin group of compounds with modifying influences of the sympathetic nervous system and the posterior pituitary hormone, oxytocin. The three major classes of medication used to prevent or suppress premature labor are: (1) Intravenous alcohol, (2) Sympathomimetic beta adrenergic compounds (Isoxuprine, Terbutaline, Ritodrine), and (3) Prostaglandin inhibitors (Indomethacin, etc.)

The use of intravenous alcohol has recently been associated with adverse fetal effects including decreased cardiac contractility in sheep, abnormal bone marrow morphology, fetal metabolic acidosis, and a tendency for muscle hypotonia.[1] These reports together with an effectiveness rate slightly lower than other treatment modalities have decreased the popularity of this method of management in many centers in recent years.

Although the early reports were enthusiastic, the finding of premature closure of the ductus arteriosus with the use of prostaglandin inhibitors has essentially relegated this modality to the research laboratory at the present time.[1, 2]

Currently the most popular methods in use revolve around the use of Beta adrenergic agonists to halt premature labor. Due to maternal cardiovascular side effects the dosage tolerated is limited in many instances and currently the thrust of clinical research is to formulate compounds with an increased uterine specificity by limiting its activity to the so-called Beta-2 (uterine) receptors and not significantly affect the Beta-1 receptors (heart). By stimulating the Beta-2 receptors the tendency of the uterus to contract is reduced.[3]

Many problems exist in both investigating the problem of premature labor as well as interpreting the work that has been done in this area. The

Table I

CONDITIONS ASSOCIATED WITH
AN INCREASED RISK
OF PREMATURE LABOR

Premature Rupture of the Membranes
Multiple Gestation
Some Uterine Anomalies
Maternal Febrile Illnesses
(Especially Pyelonephritis)
Polyhydramnios

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* Presented at the Second Annual Meeting of the South Carolina Perinatal Association, Columbia, S. C., January 1979.

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first problem that is encountered is that the diagnosis of early latent phase labor is frequently difficult to make. In studies with very lenient criteria the success rate of any modality will be good because many of the patients will be in false labor and would have ceased contractions spontaneously. Secondly, the beneficial effects of bed rest, lateral recumbent positioning, and fluid administration have been documented in the past.[4]

In summary, premature delivery is a major problem in the practice of obstetrics and the mortality and morbidity that result from prematurity are indeed staggering. The future holds a promise of a better understanding of the underlying physiology and pathophysiology and, therefore, better treatment methods.

The second major group of low birthweight infants are those that may or may not be premature at birth, but are small for their gestational age. The realization that this problem occurs in two to eight percent of the population and yet accounts for a much larger percentage of perinatal deaths, as well as resulting in some degree of learning impairment and/or significant EEG abnormalities in as many as 40 to 50% of the neonates justifies intensive efforts to improve the prenatal, intrapartum, and neonatal management of these pregnancies and newborns. Furthermore, the fact that we diagnose prenatally only 60% of the cases, should demand that we re-evaluate our methods of antenatal management.[5]

Table II is only a partial list of the various names by which this clinical syndrome has been referred to in the past. It is apparent that many of these terms reflect the individual authors bias regarding the underlying pathophysiological mechanisms responsible for the abnormality. At the present time there is still disagreement as to the etiology, morbidity, management, and nomenclature of the pregnancy characterized by inadequate fetal growth. Some of the more frequent maternal disease states associated with abnormal fetal growth are shown in Table III.

Dr. Frederick Battaglia has recently reviewed the pertinent factors that influence the growth of the human fetus.[6] These can largely be divided into two broad categories, 1) Intrinsic abnormalities within the fetus and/or placenta, and 2) Limitation of the entry of substrate into the fetus.

Included in the first category should be the chromosomal abnormalities, particularly the trisomies, intrauterine infections such as CMV, toxoplasmosis, possibly Herpes viremia, and syndromes of various congenital anomalies associated with short stature and growth retardation such as the Silver-Russel syndrome. It should be noted that significant placental abnormalities such as villitis and vascular abnormalities are frequently found in this group.

There is a great deal of indirect evidence that the major problems in the vast majority of SGA pregnancies is indeed limitation of substrate supply to the fetus. This problem may arise due to any of the following problems: 1) Alteration in maternal substrate concentration, 2) Alteration in placental perfusion, 3) Alteration in placental permeability, and 4) Alteration in placental metabolism.

In an analysis of the results of intensive evaluation of the placentas from SGA pregnancies, Altschuler et al noted a variety of gross and histologic abnormalities[6] (Table IV). If we accept that interference with substrate availability to the fetus is the common pathophysiologic mechanism by which the syndrome is created, it is not surprising that 40% of the placentas studied

Table II

SYNONYMS FOR INTRAUTERINE GROWTH RETARDATION

Small for Dates
Small for Gestational Age
Fetal Malnutrition
Pseudoprematurity
Dysmaturity
Placental Insufficiency
Chronic Fetal Distress
Fetal Microsomia
Primordialis
Intrauterine Dwarfism

Table III

MATERNAL DISEASE STATES ASSOCIATED WITH ABNORMAL FETAL GROWTH

Maternal Vascular Disease
Chronic Antepartum Hemorrhage
Cyanotic Congenital Heart Disease
Hemoglobinopathies

Table IV

THE PLACENTAL PATHOLOGY OF 63 SGA INFANTS[6]

Primary Diagnoses	*No.*	*%*
Placental "circulatory disturbances"	27	43
Placental infections		
Villitis, unknown	15	24
Villitis, cytomegalovirus	2	3
Chorioamnionitis	1	1
Abnormal placentation, excluding single umbilical artery	5	8
Placentas associated with congenital anomalies (2 of which are "normal")	7	11
"Normal" placentas	3	5
	63	100%

[6] Altshuler, Russell, and Ermocilla
February 1, 1975
Am. J. Obstet. Gynecol.

had significant vascular abnormalities. It is of interest that 27% of the placentas studied showed histopathologic evidence of villitis and implies, at least, that intrauterine infections could possibly be a significant cause of this syndrome.

One possible cause of insufficient substrate availability to the fetus is decreased substrate concentration in the maternal blood. With the exception of glucose, few studies have provided quantitative information regarding the quantity of substrate delivered to the fetus proportional to maternal blood concentration of substrate. In all likelihood, the relationship of fetal extraction of substrate to maternal blood substrate concentration will be complex. Extensive clinical trials of dietary supplementation in New York, Bogota, Guatemala, and Montreal have provided some conflicting data, but have shown the relatively poor results of routine protein supplementation alone, whereas caloric plus protein supplementation is beneficial to some degree as measured by increasing birthweight of the offspring.[7]

DIAGNOSIS AND ANTEPARTUM MANAGEMENT

The diagnosis of intrauterine growth retardation is usually difficult to make. Several essential items are beneficial in establishing the diagnosis: 1) High index of suspicion, 2) History or physical evidence of associated disease, 3) Serial measurements of the uterine fundus, and 4) Serial ultrasound evaluation of the biparietal diameter, head/abdominal circumference, and total intrauterine volume.

One philosophy of management of pregnancies with this complication is to deliver the fetus at fetal pulmonary maturity in order to remove the fetus from the delerious environment as soon as possible.[8] This may be an overly aggressive approach and is by no means a universally accepted principal. Many centers feel that in the absence of signs of utero placental insufficiency, pregnancy can be continued until spontaneous labor occurs. Due to the studies available showing major degrees of learning disability as well as poor coordination and speech problems in infants delivered after awaiting spontaneous labor, our institution, as well as many others, has adopted the more aggressive approach. The future holds the possibility of developing ways to improve the intrauterine environment without resorting to early delivery.

At the present time two types of intrauterine growth retardation are thought to exist. The first and most common is referred to as asymmetric IUGR and is thought to be caused by preferential shunting of blood flow to the fetal brain and consequently results in the so-called "brain sparing effect." For this reason measurements of the biparietal diameter alone will not diagnose many cases of intrauterine growth retardation. The second major type is felt to be more severe in degree and results in symmetric retardation of growth of the head and body. A higher incidence of fetal anomalies are found in this group.

The use of the total intrauterine volume is helpful and is based on the premise that the earliest detectable sign of intrauterine growth retardation in many cases is a decrease in amniotic fluid volume.[5] The hourly fetal urinary production rate (HFUPR) has been measured by ultrasound and has been found to be reduced in IUGR pregnancies.[8] This method also requires knowledge of the gestational age, however, because the calculated TIUV is plotted against gestational age.

In our institution, if the diagnosis of IUGR is *unequivocally* made after the 32nd week of gestation, an amniocentesis for fetal pulmonary maturity is performed and the infant is delivered if fetal pulmonary maturity is documented. In the borderline or immature category, the patient is instructed in a nutritious diet, increased bed rest, and no smoking. Biophysical (nonstress test, contraction stress test, fetal breathing determination) and/or biochemical (estriol) tests of placental function are performed serially. Every effort is made to delay delivery until evidence is established that the infant will do well in the nursery. Occasionally all parameters point to impending fetal demise if the infant is not delivered, and in this case we feel that the best chance the infant has is in the newborn intensive care unit. Fortunately, this latter case is very rare.

During labor both the premature and the small for gestational age fetus are monitored very closely. Extensive use of devices to monitor fetal cardiac rate (both doppler and scalp electrode), uterine contractions, and fetal scalp blood pH determinations make up the foundation of good intrapartum care. Prolonged labor is avoided and cesarean section is liberally used in cases of failure to progress, malpresentation (especially breech), and extreme cervical unfavorability. Cesarean section rates in the range of 40% in pregnancies with small for gestational age infants have been reported.[9]

The neonatal care of these infants is extremely demanding and requires a unique type of expertise. The details of this management will not be addressed in this monograph. Suffice it to say that these infants are very sensitive to hypoxia, hypothermia, and trauma. They are also extremely prone to develop hypoglycemia and hypocalcemia.

SUMMARY

The problem of the low birth weight infant is probably the single most important problem facing obstetrics and neonatology today. No efforts should be spared to uncover additional information relative to the cause of premature delivery and inadequate intrauterine nutrition. In this way perhaps we can not only find a better way to treat the problems once they have occurred, but hopefully to prevent them in the future. Prevention of intrauterine growth retardation must rest on assurance of adequate prenatal nutrition through prenatal nutrition education, as well as meticulous antenatal care and correct management of coexistent maternal disease. A firm commitment should be made to limit smoking and ethanol consumption in pregnant women. Careful prenatal evaluation to establish the gestational age of the pregnancy, as well as employing all available methods to assure acceptable fetal growth and development form the basis of good prenatal care of all gravidas. The use of newer modalities such as serial diagnostic ultrasound evaluations during pregnancy offer a great ray of hope. ☐

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UTILIZATION OF PRN DRUGS IN LONG-TERM CARE FACILITIES IN SOUTH CAROLINA*

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A. M. SEAR, Ph.D.

Approximately 9,025 of the older population of South Carolina live in 138 skilled and intermediate nursing care facilities licensed by the state. Most of these nursing homes do not have physician services daily. Except in emergencies or unusual circumstances, physicians attend and review the care of their nursing home patients every 30 days in skilled nursing care facilities and every 60 days in intermediate care homes.

As a result of continuing advances in medical science and drug technology, drug therapy is the most frequently employed treatment modality in medical care. This is especially true for patients in nursing homes. There is very little information available on drug therapy and utilization in long-term care facilities.

This report identifies (1) the percent utilization of prescribed PRN drugs, (2) the mean number of PRN drugs prescribed per patient, and (3) categorizes PRN drugs as to percent prescribed and percent utilized.

RESEARCH METHOD

The population studied came from a representative sample of all patients residing in 138 state-licensed skilled and intermediate care nursing homes in South Carolina. There is a wide variation in the number of beds per nursing home, with a range of beds from 10-275 and only three nursing homes with over 150 beds. Therefore, a stratified single stage cluster random sampling technique of nursing homes was used to assure that each skilled and intermediate nursing home had an ascertainable chance of being in the sample and to make certain that an adequate patient sample was drawn from the nursing homes with the small number of beds. Descriptive statistics of the population base and sample studied are shown in Table I.

From the random sample selected, each patient's record was reviewed. A list of each patient's PRN drug prescription with dosage, dosing schedule, and indication for use were recorded for a 30-day physician's prescription period. The amount of each of the prescribed PRN drugs administered for a period of 60 days was recorded from the patients' medication administration sheets. Only one of the 18 homes visited did not have records of PRN drugs used in the medication administration sheet. Information on drug utilization from that home was extracted from individual patient nursing notes.

Table I

Type Home	*Number of Homes*	*Homes Sampled*	*Total Beds*	*Beds Sampled*	*% Beds*
Very Large	3	3	701	240	34.2
Large	21	2	2,508	229	9.1
Medium	47	5	3,535	362	10.3
Small	67	8	2,281	277	12.1
Total	138	18	9,025	1,108	

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RESULTS

Results of the study are shown in Table II.

Table II

Tabulated Results	
Number of Facilities Sampled	18
Number of Facilities Studied	18
Number of Beds Sampled	1,108
PRN Drugs Used 1 or More Times in 60 Days	1,444 (44.8%)
PRN Drugs Not Used in 60 Days	1,777 (55.2%)
Range of Prescriptions/Patient	0-13

Number of prescriptions per patient and their frequency of distribution are shown in Table III. The mean number of PRN prescriptions per patient within the 30-day review was 3.

Table III

Number of Prescriptions Per Patient	*Frequency*	*Percent of Total Patients Studied*
0	88	7.94
1	182	16.4
2	248	22.4
3	237	21.4
4	162	14.6
5	89	8.03
6	43	3.88
7	22	1.98
8	19	1.71
9	9	0.722
10	3	0.271
11	1	0.09
12	3	0.271
13	2	0.181

Categories of PRN drugs prescribed, their percentage of the total PRN drugs prescribed and their percentages of USE and NON USE are shown in Table IV.

A total of 3,221 PRN drugs were prescribed for 1,108 patients during a 30-day period. Of the 3,221 PRN drugs ordered by physicians, 1,444 PRN drugs were administered one or more times to the patients for whom they were ordered. Therefore, there was a 44.8 percent utilization of PRN prescribed drugs one or more times during a 60-day period following the date of each drug order. There was a 55.2 percent of Non Use of PRN drugs ordered by physicians during the 60 days after the drug had been ordered or made available.

The varieties of drugs prescribed for PRN use fell into 15 different categories based on the therapeutic effects of the drugs. The category of drugs prescribed most frequently was analgesics (28.9 percent of all PRN drugs prescribed) followed by the laxatives (22 percent of all PRN drugs ordered). The least prescribed category as shown in this study was ophthalmic medications. In the analgesic category, 49.3 percent of the PRN drugs were used at least once within 60 days of order, while 50.7 percent were not used even once. For the laxatives, 48.4 percent were used and 51.6 percent were not used within 60 days. The category of PRN drugs which were most used was the hypnotics. Although hypnotics constituted only 4.2 percent of all the PRN drugs ordered, 59.7 percent of the drugs prescribed were used during the 60-day study period. The most unused drugs ordered were in the category of the antidiarrheals. This category constituted only 3.4 percent of all the PRN drugs prescribed; however, 78 percent of them were not used within 60 days after their order.

DISCUSSION

The results of this study suggest that there is an over prescribing of PRN drugs in skilled and intermediate nursing homes in South Carolina. Since 55.2 percent of 3,221 PRN drugs prescribed were not used one or more times during the 60 days following each of the prescriptions, this implies that the drugs were either not needed or not requested by the patient. It is reasonable to infer that since these drugs were actually supplied as prescribed, the cost of the drugs and the fees for dispensing were paid through private or governmental sources. Studies[1] have shown that by far the greater percentage of drug cost in long-term care facilities is covered by governmental funding. In a world where resources are scarce and the rapid rise in the cost of medical care has been an alarming and insurmountable problem, it cannot be overemphasized that tailoring medical care to actual needs of the patient will help contain health care cost. A recent publication by the Department of Health, Education and Welfare[2] states that the principal component of long-term care is chemotherapy and the summary of the findings show that prescribing patterns raise many questions and serious concerns about drug therapy and pharmaceutical services requirements as they affect the quality of care provided in skilled nursing homes.

Table IV

Drug Categories	*Number Prescribed*	*% of Total*	*Amount Used*	*% Used*	*% Not Used*
Analgesics	916	28.9	452	49.3	50.7
Laxatives	709	22.0	343	48.4	51.6
Hypnotics	134	4.2	80	59.7	40.3
Stool Softeners	107	3.3	45	42.1	57.9
Expectorants and Antitussives	166	5.2	62	37.3	62.7
Antihistamines	85	2.6	34	40.0	60.0
Topical Medications	178	5.5	87	48.8	51.2
Antidiarrheals	109	3.4	24	22.0	78.0
Antacids	113	3.5	47	41.6	58.4
Antinausants	132	4.1	45	34.2	65.8
Ophthalmic Medications	39	1.2	18	46.2	53.8
Anti-Angina	76	2.4	37	48.2	51.8
Antianxiety and Sedatives	175	5.4	65	37.1	62.9
Antipsychotics	86	2.7	37	43.0	57.0
Others (antihemorroids, diuretics, lozenges, etc.)	196	6.1	68	34.7	65.3

However, there are reasons that may warrant the over prescription of drugs for nursing home facilities. The absence of a physician for extended periods of time in many of the nursing homes necessitates the prescribing of many common PRN prescribed drugs in order for the nurse to properly handle such common geriatric problems as diarrhea, psychomatic pain, minor skin disorders, etc. Nevertheless, the study does point up the need to take a close look at the practice habits of physician prescribing and patient management in skilled and intermediate nursing facilities.

More important is the need to look at the drug dispensing patterns of the nursing home and the state regulations controlling the number of doses which must be dispensed. The regulations of the South Carolina Department of Social Services[3] mandate that a minimum of 30 days drug supply should be dispensed for prescriptions that call for certain categories of drugs. Most PRN drugs fall in these categories. Therefore, emphasis should first be placed upon the re-evaluation of state policies governing the administration and dispensing of PRN prescribed drugs in skilled and intermediate nursing homes where more PRN drugs are likely to be ordered than any other sector of the health care industry.

Current pharmaceutical practices recommend unit dose drugs distribution. The American Society of Hospital Pharmacists[4] cite the following distinctive elements which are basic to all unit dose systems. Medications are contained in single unit packages. They are dispensed in as ready-to-administer form as possible. For most medications, not more than a 24-hour supply of doses is delivered to or available at the patient care area at any time. Numerous studies[5] concerning unit dose drug distribution systems have been published over the past several years. These studies indicate categorically that unit dose systems, with respect to other drug distribution methods are: (1) safer for the patient, (2) more efficient and economical for the institution, and (3) a more effective method of utilizing professional resources.

The conditions of participation for the Medicare and Medicaid[6] programs require that a pharmacist be responsible for developing, coordinating and supervising all pharmaceutical services according to acceptable professional standards; that he review the drug regimen for each patient monthly and that he be accountable to the Pharmaceutical Services Committee of the facility and to the medical director and the administrator. These extended care facility regulations were enthusiastically greeted by pharmacists, but the lack of reimbursement for such services drove the pharmacists back, thus contributing substantially to the development of the "paper consultant" phenomenon in which the pharmacist-consultant of patients' records seldom did more than supply prescription medication to unseen nursing home patients. Kidder[7] documented in his study that the Medicare and Medicaid regulations that require pharmacists to review monthly the drug regimen of each skilled nursing home facility patient has the potential for savings in these programs of $3.2 to $37.2 million a year.

Brodie et al.[8] indicate that a means of developing the existing drug use profile for both individual patients and the nursing home facility can be achieved if the pharmacist records the number of medicines consumed and the number of daily doses administered to each patient at the time of the monthly drug regimen review. Once these profiles have been established, changes in utilization and costs on a before-and-after basis are evaluated. Their study showed that this mechanism for acquiring drug use data at the time of the monthly patient review had been tested and found practical. Data acquired in one facility of 55 beds for 13 months during which this prescribed model had been established, show that the number of drugs administered daily was reduced from 6-8 to 4-6; the cost equivalent of this reduction was 40 cents per patient day. The savings were attributed to effective automatic stop order policy and careful review of patients' medication profiles by the pharmacist.

One of the key objectives of monthly drug regimen reviews in nursing home facilities is the reduction, subject to the approval of attending physicians, in unwarranted and unnecessary drugs. Such reduction not only saves unnecessary cost, but also decreases patient exposure to possible drug-drug interaction. A study conducted by Brown[9] showed that 27 residents in homes for the elderly were potential drug-drug interactors, and that these subjects had twice as many drug products and three times as many PRN drugs prescribed for them as did subjects without the potential for interaction.

Emphasis should be placed upon better use of the pharmacist in the management of the health care provided in long-term care facilities. By development of an active drug use profile for both individual patients and nursing homes, and through the application of unit dose dispensing of medications, pharmaceutical services can be more appropriately used. Improved pharmaceutical services in nursing home facilities can provide both an increased savings in cost and improved quality of medical care provided.

SUMMARY

A study of the utilization of PRN drugs in long-term care facilities in South Carolina was conducted. Fifty-five percent of the PRN prescribed drugs were not utilized within a 60-day period after being made available. The mean number of PRN prescriptions per patient within the 30 days of physican orders was 3. Categories of PRN drugs prescribed, their percentages of the total PRN drugs prescribed and their percentage of *use* and *non use* were shown. Comments are made as to the possible implications of these findings and suggested methods of improvement of the utilization of drugs in nursing homes in South Carolina. ☐

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Editorials

THOMAS A. ROE FOUNDATION ANNOUNCES AWARD FOR SCIENTIFIC ARTICLES WRITTEN BY PRACTICING PHYSICIANS

The Thomas A. Roe Foundation, a private Greenville foundation, has announced a $5,000 award for the best scientific article published in *The Journal* during 1980 by a South Carolina practicing physician.

The award was conceived by Dr. William H. Hunter, a member of the board of trustees of the foundation, and was announced jointly by Mr. Thomas A. Roe, the foundation's chairman, and by Dr. Harrison L. Peeples, SCMA president. The award will be administered by the South Carolina Institute for Medical Education and Research.

Eligible for the award will be all South Carolina physicians *other* than those identified as full-time faculty members. Work done during residency, fellowship, or other full-time training at an academic institution will not be considered for the award. *The purpose of the award is to encourage scientific contributions by practicing physicians.* All scientific articles in *The Journal* by practicing physicians will be considered for the award, which will be decided by a panel of judges.

Within recent memory, South Carolina practitioners have made a number of contributions to medicine, such as well-known advances in hip and prostate surgery. Nevertheless, a statistical analysis of publications in *The Journal* demonstrated a marked decline in authorship by practicing physicians. This decline was attributed to the growth of full-time faculties and to the increasing cost and specialization of research. Yet it is felt that practicing physicians at the "firing line" still have much to contribute, and this generous award may serve as an additional incentive to do so.

— CSB

THE UMPIRE'S CALL

Pittsburgh survived to win the World Series, football bowl bids are all decided, and the basketball season is well on its way. All three are vastly different team sports, but all have in common the ever present official, be he called umpire, judge or referee. Officiating is a necessity when opponents face each other in athletic team events in order to maintain fairness and enforce the rules of play — said rules having been developed by the various leagues or conferences before the season's start. To further assure fairness, designation of officials is agreed upon by the member teams in a league or conference, and when teams from different leagues or conferences face each other, each has representative officials.

Even though all efforts go to maintain fairness, at times "bad calls" are made by officials. The instant replay must be the bane of an official's existence. Even when a "bad call" is made, it is usually an honest mistake and the instant replay camera has had an angle advantage which the official did not have. Regardless of the fact that T.V. technology might show an officiating error, the call still stands because there is the need for an immediate, on the spot ruling which is official.

By now, I am sure the reader is saying to himself — "I know all that. What's the point?" The point is that the physician-patient team finds itself daily affected by official rulings in our dealings with third party payers and government agencies. One might look on it as the physician-patient team being in one league and the third party payers and government agency folks being in another league. Opposition situations invariably arise on occasion. Early in such a contest, it becomes apparent that their league, for the most part, furnishes all the officials, has made all the rules and continues to make rules affecting the contest, not only while it is going on but retroactively after it is over. We and our patients seem to have very little representation. Their officials make bad decisions, just as do the athletic officials, and they stand because they are official. Rarely is appeal or redress available to us, and even when it is, the official action still is in their hands. From the outset, their league has made the rules essentially unilaterally with little, if any, opportunity for our league to contribute.

On the surface, it all looks fairly bleak for our team and one might say, "What's the use?" In the socioeconomic arena with third party and government agencies, the physician-patient team must look to the physicians through organization as the players, while the patients who have essentially no organizational capacity can only act as fans. Opportunities to insure fairness for the physician-patient team are few, but they are there, and they can be strengthened. Of primary importance is a strong, representative, unified league and membership in the South Carolina Medical Association affords for the physician that strength and representation which will ultimately be beneficial to his patient as well. Unfortunately, many of the doctors who complain the most about the unfairness of the system have not availed themselves of membership in the SCMA. As members, they could add greatly to SCMA strength and influence.

Of equal importance is the use of the energies of the individual SCMA members through service on the various committees of the Association. All physicians in the state owe a debt of gratitude to those SCMA members who give their time and energy to ad hoc and standing committee service. Some of these committees do have input with the third party and government agency folk — to name a few: Insurance Peer Review Committee, Legislative Committee, Committee on HSAs and the many liaison committees that maintain communication with such groups as the Department of Social Services, S. C. Hospital Association, Blue Cross and Blue Shield, the South Carolina Bar Association, etc.

This is the month for annual dues billing to all physicians in the state. If you are already a member of the SCMA team, your support is a great asset. Grass-roots solicitation is probably the only means of effectively increasing SCMA membership, so if you have been warming the bench, now is the time to get into the game by encouraging your non-member colleagues to join SCMA. If you are interested in a stronger personal role on the team, let your committee interests be known — you can bet the coach will let you play.

Our team accomplishments will never appear spectacular; therefore, there will never be much cheering from our "patient-fans." If we fail, however, one can bet the boos will be forthcoming. Help us field the best team possible through strength of numbers and energetic participation.

E. Mims Mobley, Jr., M.D.
Councilor, Third District
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SEASON'S GREETINGS!!!

The Editor, Editorial Board, and staff of *The Journal of the South Carolina Medical Association* wish for each of you a joyous Christmas Season, and a happy, prosperous and peaceful New Year.

President's Page

THE "PROFESSIONAL CLUB OF MEDICINE"

SCMA and the AMA accomplish much for the benefit of the medical profession and for the patients that we serve. This is made possible by dues paid by our members. Since there is no clear distinction between the physicians who are members and those who are not, we wind up representing all the doctors whether we or they like it or not.

Dr. Jim Sammons, Executive Vice President of the AMA, states in a recent address that every "doctor ought to be a part of the Federation — the 'professional club of medicine' — and we ought to be proud of it." There are far too many getting a free ride and far too many who would sit back and "let George do it." Well, George can't do it by himself because . . . "the problems are getting worse, not better, more acute not resolved; and if we want to protect the public we serve, and if we want to protect the physician population in this country," we'd better get our act together — all of us.

Dr. Sam Nixon, President-Elect of AAFP, following his recent election, told the *Medical Tribune*, "I urge every physician to join the AMA," he said. "I think the Academy should occupy a fair amount of space in the 'House of Medicine.' "

He rejected the contention of some physicians that the AMA public image is smeared so badly that its effectiveness in Congress is impeded. "The AMA didn't do it," Dr. Nixon says. "That image was created by people not connected with medicine. They set up a straw man, then attacked it, and news reports of that spilled back to the physician who holds it against the AMA." Who are these people setting up the "straw man"? "Proponents of destroying the present pleuralistic system of offering health care," Dr. Nixon said.

Last year, there were a little over 400,000 doctors of medicine living in the United States, around 300,000 delivering some form of patient care on a daily basis. One hundred fifty-five thousand of those physicians were active members of AMA. That's not near enough. Doctors can afford to pay the dues, and they may soon learn that these dues represent the best insurance premium that they pay. It's time for us to individually ask our colleagues to join the medical society; let them know we want them and need them.

The prime requisites of a medical organization are involvement, furtherance of the art of science and medicine, and the betterment of public health. We want to bring all physicians in with us, not only to help pay for it, but to help work at fulfilling our mission.

You who take the time to read these pages know the issues confronting us, and you know that we must be involved at the county, state and national level, without any more verbiage from me. I urge that you take a little more of your time to point out to your fellow physicians the need for the "Professional Club of Medicine" and the need for involvement. We must work out in conferences among ourselves an acceptable "bottom line" and make a common commitment to live within it.

At the Holiday Season, my thoughts turn gratefully to those who have made our progress possible. It is in this spirit that I say THANK YOU AND BEST WISHES FOR THE HOLIDAYS AND A HAPPY NEW YEAR.

Harrison L. Peeples, M.D.,
President

"Better Health Through Health Education"

SOUTH CAROLINA MEDICAL ASSOCIATION

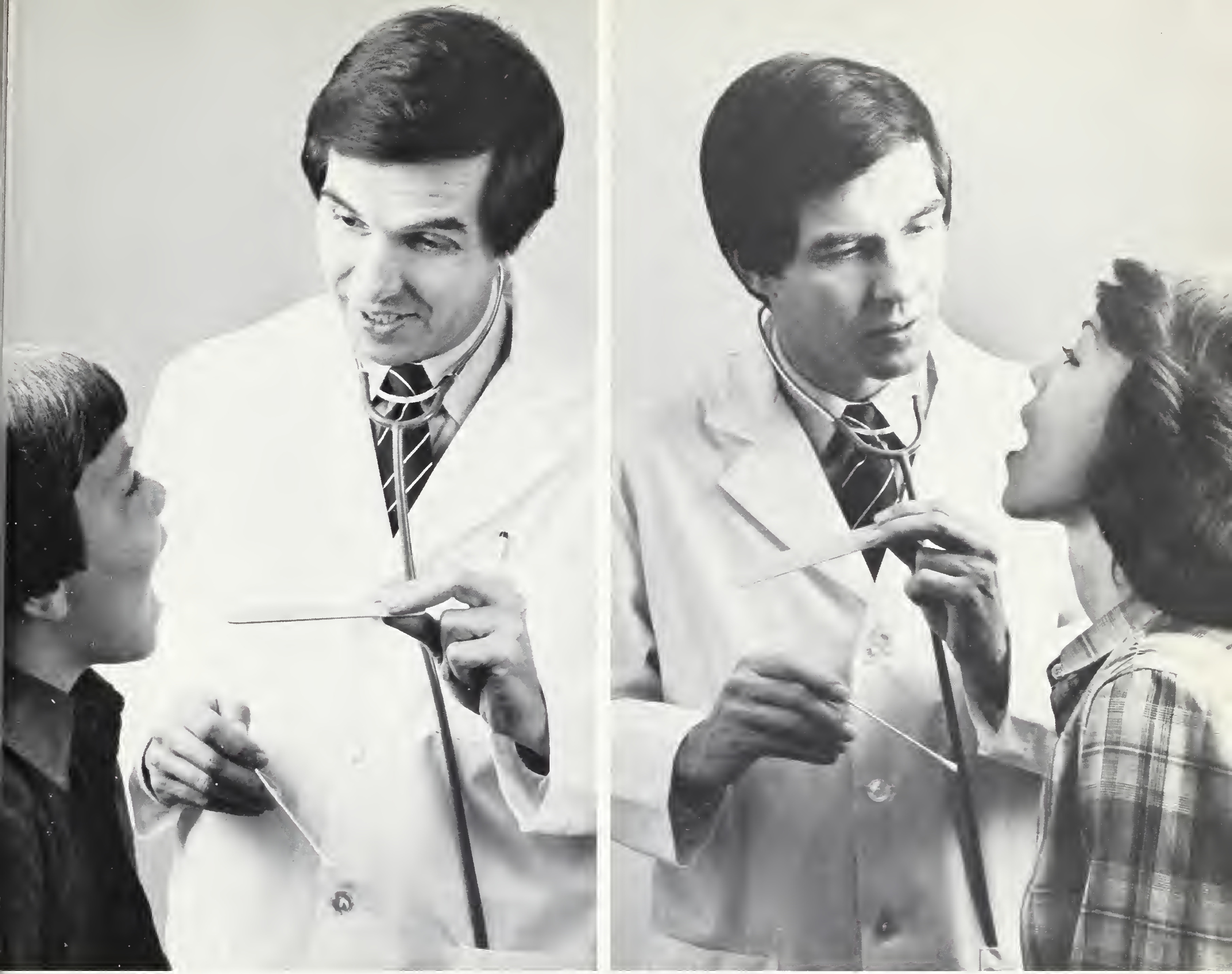
AUXILIARY

COLUMBIA MEDICAL SOCIETY AUXILIARY

A tradition began four years ago for the Columbia Medical Society Auxiliary. The AMA-ERF co-chairmen, Mrs. Richard K. Truluck, Jr. and Mrs. Ralph Owings, hit upon the idea of utilizing the artistic talents of our auxiliary members and creating our very own Christmas card. This year's card has been designed by Helen Macdonald. She is the wife of Dr. Roderick Macdonald, dean of the University of South Carolina Medical School. Helen's lovely and appropriate card has enabled us to send a personal holiday greeting to all members of the Columbia Medical Society.

Mrs. Jeff Brooker
Mrs. Dan Paysinger
Co-Chairmen, AMA-ERF,
Columbia Auxiliary

The South Carolina Medical Association Auxiliary wishes each of you a joyous and blessed holiday.



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